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Editorials.

ANTHRAX IN THE HORSE.

By means of a short communication to the Central Veterinary Society, which we reproduce in this issue of the Veterinary Journal, Mr. Hunting has drawn our attention to the unsuspected frequency of anthrax occurring amongst horses in London. A very important feature is the fact that diagnosis in each of the cases with which he has come into contact has been made only on *post-mortem* examination, the horses in every instance having been treated for colic. That being so, it is highly probable that many other fatal cases of colic on which no *post-mortem* examination was made may also have been anthrax. The serious side of the matter is, of course, increased by the infectivity of anthrax for man, and the ease with which a knacker or other person might become affected in cutting up the carcase. Of course, if definite diagnosis could be made *inter vitem*, it would not materially affect the patient's chance of recovery, but it would certainly make a difference in the treatment of the discharges, and ultimately of the carcase.

In connection with diagnosis, Mr. Hunting suggested the possibility that the more frequent use of the clinical thermometer might assist in differentiating the abdominal form of anthrax from so-called colic, since most text-books assert that there is always a very high temperature in anthrax. Friedberger and Fröhner, in their "Veterinary Pathology," state that the first symptom of anthrax in the horse is "high fever, ranging from 103° to 107° F., or more." Our personal experience of anthrax in the horse is very limited, but in two cases diagnosed as colic circumstances permitted of the temperatures being taken frequently during the thirty-six to forty-eight hours they were ill.

In neither case did the temperature rise to more than 102° F., until about two hours before death, when it went up somewhat rapidly, and pain diminished. A definite diagnosis of anthrax was made after death. One frequently meets with a similar temperature in colic, so that it seems somewhat doubtful whether the thermometer would be of much service in differential diagnosis.

Mr. Hunting also raised another important question from the economic aspect. It is believed that infection in these cases takes place through contaminated food, and Mr. Hunting asks, "What would you do with the food?" No doubt most veterinary surgeons would immediately reply that it should be either destroyed or submitted to some efficient cooking process, as boiling or prolonged steaming. But, judging from Mr. Hunting's cases, this procedure would appear to be unnecessary. The whole question is complex and full of difficulties, and it would be very instructive if practitioners would record their observations on this point, and so assist in the solution of the question.

THE THIRD INTERIM REPORT OF THE ROYAL COMMISSION ON HUMAN AND ANIMAL TUBERCULOSIS.

THE above interim report has been issued, and a fairly full abstract will be found elsewhere in this issue. The report is accompanied by an appendix, written by Dr. Griffith, indicating the methods of procedure employed by the Commission in their inoculation and feeding experiments with milk and fæces of naturally infected tuberculous cows. The appendix also includes full details of the *post-mortem* examination of all the cattle naturally affected, and of the guinea-pigs and swine experimented with. The report embodies the conclusions which may be drawn from the experiments, and the whole publication is full of very instructive information.

Of the six cows used for the purposes of these experiments, three only were clinically tuberculous, while all gave a positive reaction to tuberculin. None of them, however, showed any clinical signs of tuberculosis of the udder. In spite of the latter fact, it was clearly shown that the milk of the clinically affected

animals, drawn in such a way as to avoid the possibility of external contamination, contained virulent tubercle bacilli. Very careful *post-mortem* examination of the udders of these cows revealed in one quarter of the udder of one cow four small tubercular nodules, which could not possibly have been detected during life. No lesions whatever were discoverable in the udders of the other two. In all three cases the fæces contained large numbers of virulent organisms. Hence the very important announcement that the milk of such cows (*i.e.*, those obviously tuberculous, even though the mammary glands are not affected) must be considered dangerous for human beings.

The enormous number of virulent tubercle bacilli that may be present in a small amount of fæces of clinically affected cows was illustrated in a remarkable degree by the Commission. One gramme (fifteen grains) of fæces was added to two litres (over three-and-a-half pints) of sterilized water and two guinea-pigs were inoculated intraperitoneally, one with one cubic centimetre and the other with two cubic centimetres of the mixture. Both developed severe general tuberculosis. We think it would be quite impossible to find a stronger argument for insisting by law on cleanly methods of milk production.

The fæces of two of the remaining three cattle contained virulent material, and as we know milk is so often contaminated with fæcal matter, milk taken under ordinary conditions from such animals can scarcely be regarded as free from danger.

The most important point, however, is the fact that in any attempt to control or prevent the supply of tuberculous milk ALL clinically affected cows must be eliminated from dairy herds, and not simply those with tubercular mastitis.

In the article by Professor Woodruff, which will be found elsewhere in this issue, much useful information and many valuable suggestions concerning the control of the milk supply are lucidly put forward. We cannot possibly continue long under the present conditions, and we trust that the Milk Bill to be shortly introduced by Mr. John Burns will tackle the whole question on broad lines, and, while avoiding any semblance of injustice to the milk producers, at the same time safeguard the public from the dangers of this living poison.

General Articles.

MILK AS A SOURCE OF INFECTION IN TUBERCULOSIS.[1]

BY H. A. WOODRUFF, M.R.C.V.S., PROFESSOR IN THE ROYAL VETERINARY COLLEGE, LONDON.

My subject is that of infection of the human subject with tuberculosis from cows' milk. It may be well at the commencement of my address to say that absolute demonstrable proof of the proposition that human beings are infected with tuberculosis by means of milk from cows the subject of tuberculosis is in the nature of the case impossible, but it will be my endeavour to show that the great weight of evidence points to the possibility—nay, more, the probability—of infection. Furthermore, notwithstanding our boasted advance in sanitary science and preventive medicine, the great bulk of our population is not protected against this danger. It is unnecessary for me to spend much time in emphasizing the value of milk as a food; it is part of the common knowledge of life that milk is in itself a complete food, but it may be pointed out that those members of the community who consume milk as their staple, and often, indeed, sole diet, are the most susceptible to disease. In milk we have a food which, besides being of almost universal use in ordinary diet, constitutes the chief food, and that frequently in an uncooked state, of a large proportion of the infants under one year old. One of the most serious questions for public health authorities, and one which intimately concerns the welfare and prosperity of the nation, is that of infant mortality. That out of every 1,000 children born in our towns from 130 to 150 should die under one year old[2] is a standing menace to our national well-being, and the only rift in the cloud is to be found in the fact that medical officers of health, public health authorities and municipal councils are alive to the danger, and are energetically working for improvement. How great is the danger may be understood from the following quotations from Dr. Newman's book: "In 1905 there was a loss to the nation of 120,000 dead infants in England and Wales alone; that is to say, one quarter of the total deaths every year is of children under the age of twelve months."[3] In speaking of preventive methods, Dr. Newman says: "There is needed increased control of the milk supply. We have seen that infant mortality, and particularly fatal diarrhœa, chiefly affects infants fed on condensed milk and cows' milk, and only in the smallest degree those who are breast-fed."[4] Further, speaking of milk production: "The importance of commencing the control of milk at the beginning can scarcely be overestimated. If the cow be diseased or unclean, nothing can correct the unsatisfactory character of the milk."[5]

But milk is also the especial diet of invalids, in whom the natural defensive forces of the body are weak; and what advice more common to the suspect or early tubercular patient than the direction to drink plenty of new milk—sound enough advice if the milk be pure, but

[1] Paper read at the National Conference on Tuberculosis, London, February, 1909.
[2] See "Infant Mortality," by George Newman, M.D., Table, p. 22.
[3] "Infant Mortality," Newman, p. 2.
[4] *Ibid.*, p. 318.
[5] *Ibid.*, p. 321.

what an irony if the milk, containing bovine tubercle bacilli, be itself infective.

And now let us consider for a moment the prevalence of bovine tuberculosis. Cattle are very commonly the subjects of this disease, and from their conditions of life milch cattle are the commonest victims, probably 30 per cent. being affected to a greater or lesser extent.

To what extent is the milk of these tuberculous animals dangerous? It has been commonly asserted that only the milk of animals with tuberculosis of the udder contains tubercle bacilli, and so is actually dangerous; and in his address to the British Congress on Tuberculosis in 1901 Sir John McFadyean said: "There is ample evidence to justify the assertion that, as a rule, the milk is not dangerous until the udder itself becomes diseased." And further he stated: "My own opinion leads me to think that about 2 per cent. of the cows in the milking herds of this country are affected with tuberculosis of the udder." The farmers and milk producers have been especially emphatic upon this point, and Mr. Primrose McConnell, B.Sc., in an address before the Royal Society of Arts in December last, said: "I think it is now generally acknowledged that there is only danger to be feared in the case of cows with tuberculous udders, and if these are removed from a herd, then the danger becomes negligible." How far this opinion minimized the actual danger is shown in part by the third interim report of the Royal Commission on Tuberculosis issued this month. The researches made by Dr. F. Griffith for the Commission show conclusively that both the milk and the fæces of cows clinically tuberculous, but without disease of the udder, may contain virulent tubercle bacilli, often in large numbers.

In their review of their experiments, the Royal Commissioners remark: "The presence of tubercle bacilli in the milk of cows clinically recognizable as tuberculous confirms the opinion we expressed in our second interim report that the milk of such cows must be considered dangerous for human beings. The experiments which we have carried out with regard to the infectivity of the fæces of tuberculous cows were dictated by knowledge of the fact that dirt of various kinds from cows and the cowshed is constantly present in milk as it reaches the consumer. Cows suffering from extensive tuberculosis of the lungs must discharge considerable numbers of bacilli from the air passages in the act of coughing, and some of the bacilli thus expelled may find their way into the milk. But our experiments indicate that the excrement of cows obviously suffering from tuberculosis of the lungs or alimentary canal must be regarded as much more dangerous than the matter discharged from the mouth or nostrils. We have found that even in the case of cows with slight tuberculous lesions tubercle bacilli in small numbers are discharged in the fæces, while, as regards cows clinically tuberculous, our experiments show that the fæces contain large numbers of living and virulent tubercle bacilli."

Even prior to this report authorities in Germany and America had realized these dangers, and in 1907 Dr. Mohler, Chief of the Pathological Division of the American Bureau of Animal Industry, reviewing the evidence which had been accumulated on this subject, wrote as follows: "That milk coming from a tuberculous udder is capable

of transmitting the infectious principle is conceded by all who have given the subject any consideration. It has been equally established that in advanced generalized tuberculosis the udder may secrete tubercle bacilli without showing any indication of being affected. Careful experiments performed by trained and eminently responsible investigators have also demonstrated beyond reasonable doubt that tubercle bacilli at certain times may be present in the milk of cows affected with tuberculosis to a degree that can be detected only by the tuberculin test, so that in a herd of cows in the various stages of tuberculosis it is to be expected that some of them will secrete tuberculous milk, which, when mixed with other cows' milk, makes the entire product dangerous."

Thus the scientific opinion of the world is united in pointing out this grave risk, greatly wider and more insidious than has been commonly suspected.

But let us see what precautions are taken to prevent this danger. Sir John McFadyean, speaking on this point, said: "If every town and rural district produced its own milk it would be a comparatively simple problem to organize and carry out a fairly efficient system of inspection of milch cows, but as the law at present stands the majority of the population cannot obtain this safeguard. With the exception of Glasgow, Manchester, and a few other places, a local authority has no power to inspect cows outside its own district, and the helpless position in which this state of the law leaves the inhabitants of London and other large towns is obvious."

Since this statement was made the L.C.C. have obtained powers to inspect cows in country cowsheds from which milk is sent into London, and the results of the first quarter's examination is startling, as we shall see later; but let us first consider the state of affairs in the town cowsheds.

In several of the large towns themselves, where inspection of cows and cowsheds prevails, the amount of disease is small, and the following figures show how valuable a safeguard regular veterinary inspection of milch cows is. In London during thirteen quarterly inspections from 1899 to 1903, the number of cows examined was never less than 3,900, and out of these the number with "actual and suspected tuberculous udders" was on the average only 0·2 per cent. In Manchester, in 1901, of 1,839 cows (in town byres) examined, only one was found to have tuberculosis of the udder (0·54 per cent.), whilst out of 1,441 cows in country cowsheds, whose milk was consumed in Manchester, fifteen were found with tuberculous udders (1·04 per cent.).

Contrasted with these figures, we have others dealing with samples of milk taken from churns sent up from the country districts to London.

At a recent meeting of the London County Council, the Public Health Committee presented a report upon the step which it had taken since July 1 under the powers granted by the London County Council (General Powers) Act, 1907. The report states:—

"A veterinary inspector has been appointed for the purposes of this Act, for the inspection of cows at farms outside London which supply milk within the county, and an arrangement has been made with the Director of the Lister Institute for the examination of samples

of milk taken under the Act. The first quarterly return of our action is now due, and for convenience we have extended the period under review to October 10, 1908.

"During this time 204 samples of milk have been taken, 60 of which were of milk carried by the London and North-Western Railway Company, 48 by the Great Eastern Railway Company, 60 by the London and South-Western Railway Company, and 36 by the Great Western Railway Company. The examination of 92 of these samples has been completed by the Lister Institute, with the result that 22 (that is, a percentage of 23·9) have proved to be tuberculous. The Council's veterinary inspector has visited twenty of the farms from which the samples were taken, and inspected the animals at nineteen of them."

As the result of clinical examinations, nine cows were at once reported by the Council's veterinary inspector to have tuberculous udders. Another cow has subsequently proved to have a tuberculous udder, making ten cows in all proved to be suffering from this disease. The owner or manager at once undertook not to send to market *in London* more milk from these cows.

These results show very clearly the conditions prevailing over the country generally, and the crying need there is for national and uniform control.

It is obviously unfair that the conditions under which a cowkeeper may conduct his business in one district should be made more rigorous than those for his competitors in a neighbouring one, and this lack of uniformity simply means that the diseased animals condemned and ejected from the dairies of a closely inspected district are sold into another, where they may be used to supply milk to the unsuspecting inhabitants without let or hindrance. In the words of the *Lancet*, commenting on this very report:—

"We make bold to consider that a 'Pure Milk' Bill such as Mr. Burns was to have introduced in the summer session, but which has not yet seen the light, provided it be wisely drafted, will prove of no less vital importance to the country than larger Government measures which do so much more for their promoters' political reputation."

The cow has been held accountable from time to time for several diseases of mankind, notably scarlet fever, diphtheria, epidemic sore throat, and epidemic diarrhœa; and typhoid infection has not uncommonly been attributed to contaminated milk. It may be said with something approaching certainty, that for typhoid and scarlet fever, and with scarcely less assurance for diphtheria, the cow is in no way accountable. These conditions, if brought about by milk, are simply due to infection of the milk somewhere between the cow and the consumer. Epidemic sore throat and diarrhœa due to milk are usually the outcome of badly kept cows and dirty cowsheds, or dirty milkers; or in a number of cases they do appear to have been due to some diseased condition of the cow's udder. However brought about, when an epidemic of any of these diseases does break out, the question of the milk supply is at once thought of, an investigation is made, and the danger is got rid of. With tuberculosis, however, the case is far different. Here we have a disease almost as common in man as in the cow; a disease insidious in its onset, believed by the man in the street

to be largely hereditary in origin, and, at anyrate, accepted as a part of the ordinary course of Nature by those who fall victims to it; and so any restrictions on a man's liberty, for example, to spit where and when he likes, are often resented; and the idea of getting rid of even those cows with tuberculous udders from our milking herds is thought to be a whim of the scientific faddist, an unwarranted hardship on the poor cowkeeper, and a question quite unworthy of urgent legislation.

In his address to the British Congress on Tuberculosis in 1901, Sir John M'Fadyean said: "The present state of the law, or rather the almost entire absence of any law dealing with tuberculous udder disease in cows is a scandal and a reproach to civilization. It hardly sounds credible, but it is a fact, that the owners of a cow in the most advanced state of tuberculosis and exhibiting the most manifest signs of udder disease may sell that cow's milk for human food as long as the sale has not been specially interdicted on the certificate of a veterinary surgeon." What seems more incredible—but is no less true—is that the statement still holds good in this present year of grace, 1909. Ladies and gentlemen, on this question our business is to *educate, educate, educate.*

But is bovine tuberculosis transmissible to man? We all remember the consternation and misgiving produced by Koch's pronouncement at the British Congress on Tuberculosis in 1901. Let me remind you of his words: "Though the important question whether man is susceptible to bovine tuberculosis *at all* is not yet absolutely decided, and will not admit of absolute decision to-day or to-morrow, one is nevertheless already at liberty to say that, if such a susceptibility really exists, the infection of human beings is but a very rare occurrence. I should estimate the extent of infection by the milk and flesh of tubercular cattle . . . . as hardly greater than that of hereditary transmission, and I therefore do not deem it advisable to take any measures against it."

Prior to this Congress in 1901, for many years it had been almost universally believed by pathologists that tuberculosis as present in the various species of animals, including man, was one and the same disease, differing slightly, if at all, only by reason of different environment—and more, that there was a possibility of infection from one animal to another and to man. That bovine tuberculosis could attack and infect mankind, causing symptoms and lesions in no wise differing from those of the commonly observed forms in human tuberculosis, was doubted by no pathologist of repute. Then came Koch's startling statement, but notwithstanding the veneration for so eminent a master, those best fitted to form an opinion were not convinced by the evidence brought forward. Our own Government at once granted a Royal Commission, appointed by the King, and that Commission, after years of patient investigation and labour, has spoken in no uncertain terms. In the second interim report, issued in 1907, the results were summarized in these words: "There can be no doubt but that in a certain number of cases the tuberculosis occurring in the human subject, especially in children, is the direct result of the introduction into the human body of the bacillus of bovine tuberculosis, and there also can be no doubt that in the majority at least of these cases the bacillus is introduced through cows' milk. Cows' milk containing bovine tubercle bacilli is clearly a cause of tuberculosis and of fatal tuberculosis in man."

Having in the course of their researches investigated some sixty cases of tuberculosis in the human subject, the Commissioners found that fourteen of them contained the bacillus of bovine tuberculosis. Again, "of the total of sixty cases, twenty-eight possessed clinical histories indicating that in them the bacillus was introduced through the alimentary canal. Of these, thirteen contained the bovine bacillus," or 46 per cent. "These facts indicate that a very large proportion of tuberculosis contracted by ingestion is due to tubercle bacilli of bovine source." A very considerable amount of disease and loss of life, especially among the young, must be attributed to the consumption of cows' milk containing tubercle bacilli." Such is the opinion of the British Royal Commission.

But what is the general scientific opinion in other countries? The International Congress on Tuberculosis, held at Washington in September last (1908), was notable for the great prominence given to this question of the relationship between human and bovine tuberculosis. Both in the official meetings of the Congress, and in a special meeting called for the purpose the matter was discussed by the experts from all the civilized countries of the world. Koch himself spoke, but with hardly the same assurance and emphasis as in London in 1901, and it may safely be said that the bulk of opinion was against him. Let me quote some important words used by Koch himself in the course of the discussion. He said: "I would again call attention to the fact that human tuberculosis is the principal point of attack, because eleven-twelfths of patients perish through this type of infection. I admit that bovine infection can occasionally occur, and I desire not to be understood in the sense as though I disregarded the endeavours to extirpate bovine tuberculosis, as far as these endeavours are dictated by agricultural and economic reasons. But I mean that it would be wrong to give to these proposals the leading place in front of the efforts to combat human tuberculosis." Further, he said: "I have not the slightest objection to the publication of an exact report of this meeting. Why should I object to the statement that I recognize the transmissibility of bovine tuberculosis to man? I have never denied it."

That, ladies and gentlemen, is ample justification for our position here to-day. We would be prepared to subscribe to every word of it, but, as Professor Sims Woodhead said in the same discussion, "As regards practical measures, we are of opinion that rather something too much than something too little should be done." I should like to draw attention to one important piece of work bearing on this part of the subject, and carried out by Calmette, of Lille. As the result of prolonged investigation he concluded that "the bacilli of the bovine type are rarely retained in the mesenteric glands of young children, but penetrate into the organism, and, on being localized in the lungs, undergo such changes with regard to their morphological and biological properties that they may be taken for the human type." In other words, he is of opinion that bovine tubercle bacilli ingested, say in milk, may pass through the mesenteric glands without leaving any trace and set up a lung lesion as the primary lesion. If this be true, the victims of milk infection are not simply to be numbered by those in whom a primary intestinal tuberculosis is found—a large enough number to justify tenfold the effort now being made for pure milk—

but by a very much greater number of cases. The analogy of glanders in horses, where infection by ingestion may set up primary lung lesions, prevents our denying the possibility of Calmette's conclusion, and the finding of the Royal Commission that tubercle bacilli can be voided in the milk without the udder being diseased still further confirms the possibility.

There is, then, in our midst a danger, insidious, silent, relentless, the extent of which we cannot with certainty estimate. Shall we in this country blind ourselves to the facts and continue living in that paradise prepared for us by the milk purveyors with the aid of Professor Koch, or shall we face the danger, grapple with it, and overcome it?

This question, ladies and gentlemen, brings me to my last point—namely, the means to be adopted whereby this danger can be overcome. There are, of course, various methods advocated, and some of these may be mentioned. The consumer may prevent any possibility of danger by boiling all the milk used, but apart from any loss of nutritive value or any possible slight ill-effects to children fed on such milk, boiled milk is to a large number of people unpalatable; there is the trouble of boiling it; and the class of people who most require protection against tubercular infection, the lower class poor, are the last to realize the need for boiling the milk they use.

Another method doomed very much at the present time is pasteurization. Now pasteurization has the advantage that it does not render milk unpalatable, whilst it does undoubtedly clean the milk and remove dirt and hair, and much foreign matter which ordinary straining does not exclude. Then, too, pasteurization if done on a large scale is cheap; the milk keeps better than if untreated, and it is easily handled in sterilized bottles without risk of contamination after leaving the dairy and before reaching the consumer. There are, however, certain conditions requisite for pasteurization to be of any real value—namely, that the milk must be submitted to a sufficiently high temperature for a sufficient length of time. Milk heated gradually up to 85° C. and kept at that temperature for three or four minutes may be considered free from living tubercle bacilli, but the public should be warned that in more than one of the large dairies where pasteurizing plant has been installed, the milk is not heated to more than 70° or 72° C., and that only for a very short time. When this is the case, pasteurization is not only useless, it is positively dangerous, for the public are brought to believe that infection with such milk is impossible, and a sense of security prevails, and, moreover, those simple saprophytes which cause souring and curdling of the milk and act as danger signals when the milk is no longer fit for food, have been killed off, so that decomposing milk may be consumed, because there is wanting any evidence of its real condition. Pasteurization, if to be advised, must be efficient and genuine, and it would be well if public health authorities had powers of supervision over those dairies where it is in vogue. But, ladies and gentlemen, these measures are mere palliatives, and, like all palliatives, they are unsatisfactory. Only one measure can really be considered efficient, and that is the eradication of clinical tuberculosis from the cows in our milking herds. The time when all the cows used for milk production shall be free from tuberculosis is not yet, but there

are certain practicable and essential measures to be taken if the danger from infected milk is to be reduced to any extent. Time forbids me to give detailed reasons for the measures advocated, but they must be briefly stated.

(1) Provision must be made for uniform and national regulation, and inspection of cows and cowsheds. A committee appointed by the Royal Institute of Public Health in 1904 to consider the question of "A Clean Milk Supply," and consisting of medical officers of health, bacteriologists, and representatives of the milk trade, in a report issued in 1906, said: "In the course of this enquiry an endeavour was made to obtain information as to the enforcement of the Order (the Dairies, Cowsheds, and Milkshops Order of 1886) in the various districts in the kingdom. One fact left but little room for doubt, viz., that in a large majority of rural districts the Order is a dead letter. Under these circumstances the committee are strongly of opinion that where the local sanitary authority is in default the obligations to enforce the Order should devolve upon the County Council, and, further, that the power to make regulations under the *Order should be made compulsory and not permissive.* A similar recommendation was made by the second Royal Commission on Tuberculosis reporting in 1898. (Recommendation 10.)

(2) It must be made incumbent on cowkeepers and dairymen to notify all diseases of the udder. It is both unsafe and unjust to allow the owner of the cow to decide which are and which are not dangerous cases, especially when we remember that even for the veterinary surgeon tuberculosis of the udder is a most difficult condition to diagnose in the early stages. The second Royal Commission (of 1898) recommended this step, whilst the second interim report of the present Commission goes beyond this and suggests a further addition to the law. Speaking of cows which may be recognized clinically to be the subjects of tuberculosis they report as follows: "Our results clearly point to measures more stringent than those at present enforced being taken to prevent the sale or consumption of such milk." The third interim report, as we have seen, still further confirms and emphasizes the necessity for this.

(3) We may then advise as our next clause: The extension of the definition of the word "disease," as used in the Dairies, Cowsheds and Milkshops Orders, so that it shall include animals clinically tuberculous.

The Royal College of Veterinary Surgeons have advised the Board of Agriculture that, in their opinion, clinically evident tuberculosis should be made a notifiable disease under the Contagious Diseases (Animals) Act of 1894.

Cows notified under this order would be dealt with in such a way that their milk would not be used for food.

(4) Instead of the present system of registration of cowkeepers a system of annual licences should be substituted.

(5) And, lastly, the powers conferred by local Acts on certain towns, as Manchester, Liverpool, Sheffield, relating to tuberculosis and milk, should be extended to the whole country, especially the power to inspect "outside" dairies from which they obtain milk.

These are among the more important items that any legislation dealing adequately with this question of the milk supply must contain, and we who know the danger of the present system must rest satisfied with nothing short of a radical, complete and national scheme for its prevention and removal.

ANTHRAX IN THE HORSE.[1]

BY WILLIAM HUNTING, F.R.C.V.S.

I WISH to state a little experience I have had during the last twelve months. I have to attend the slaughter-house every day, and I occasionally find cases of anthrax amongst horses; during the last twelve months we (my colleague and I) have found nine. The peculiarity of those cases is that not one was diagnosed by the veterinary surgeon, and no one dreamt they were cases of anthrax until the *post mortem*. I do not say that to reflect on the veterinary surgeon, because I myself do not know what the symptoms of anthrax are in the horse when he is alive. I never saw a case to my knowledge, but I have occasionally made *post mortems* on horses that I thought died of colic, and wondered what in the world the lesions I found were. I know now they were anthrax. I thought I would draw the attention of practitioners in London to the facts.

One horse was in a stud of three, another came out of a stud of four, another out of a stud of forty, another from a stud of 3,000, two from a stud of 300 at an interval of less than a month, and two from the country, where they might have contracted the disease by the contamination of cattle.

The *post-mortem* symptoms that I rely upon are these: Subcutaneous patches with dark, black hæmorrhage, very often on the shoulders and ribs—that is the first thing you see as the horse-slaughterer takes the skin off; then enlarged and darkly-stained mesenteric glands, with a large quantity of gelatinous effusion in the mesentery, and especially along the double colon. These cases were not only diagnosed by those *post-mortem* symptoms, but, thanks to my colleague, Mr. Humphrey, a microscopic corroboration was made in every case, and in some cases his examination was again verified by the Board of Agriculture.

As I said before, none of these cases was diagnosed alive, and that suggests to me that possibly there may be a good number of these cases in London that are not discovered. A very curious thing is that in not one of these cases was the food changed, the food that was supposed to affect the horses that died being continued and yet no other case happened, except on one occasion. In the case where there were two horses in 300, one occurred in October and the other in November, an interval of a fortnight. In another case a veterinary surgeon had made a diagnosis and said: "I suspect anthrax, and I wish you would look at it with me." It was anthrax, and was corroborated by the Board of Agriculture. The reason the veterinary surgeon diagnosed the case was that only ten days previously, in the same stud, he had had a case exactly like it.

[1] Read before the Central Veterinary Medical Association on January 7, 1909.

So far as I can trace any of the history of these cases, they are all alike in their symptoms. They are treated for colic, and they come in to the knacker's with the idea that the veterinary surgeon will find a twisted colon, or something of that sort. Their existence is very short. Three cases amongst them had been taken ill on the road and run in to the nearest veterinary surgeon and treated for colic. So far as the appearance of the carcase was concerned, there was a differentiation between death from anthrax and death from twisted large intestine in the amount of swelling of the abdomen. In none of our *post mortems* was the horse particularly swollen—unlike the cows and oxen which are found blown up.

In one case I looked up the owner to see the place disinfected, and he told me that the day previous to the death of the horse it had been in the stable doing nothing, and he said to his man, "Bring that horse round and take a load of stuff somewhere." The man brought the horse round, but the master did not like the look of him, and asked the man whether the horse had eaten his food during the night, and the man said he had not. The master said, "Then take him back home, as he is not fit to go out." The horse went about 50 yards and then fell down, and they could not get him up. He seemed in a good deal of pain. They sent for the ambulance, but fortunately it arrived just after the animal's death, so that the vehicle was not infected, and the horse was taken away in a horse-slaughterer's cart.

In another case the veterinary surgeon attending the stud was a friend of mine, and I rang him up on the telephone and asked him whether he knew what he had got, and he said it was twisted gut. I said, "No, anthrax." He said, "That is curious; we have had a lot of twisted guts lately, but I have not had time to make any *post mortems*."

I think it is worth while bringing this forward just to suggest that anthrax is more common in London than we suppose, and that the diagnosis is almost impossible during life; at any rate, none of these cases were diagnosed.

The books state that there is always very high temperature in cases of anthrax, and I think I am right in saying that there is not often very high temperature in "colic" until it gets near the end, and I am wondering whether the more careful use of the thermometer in cases of so-called colic would help us in our diagnosis of the disease.

Now as to prevention. Imagine that it was the food that produced the disease, what would you do with the food? If in the cases I have collected you had destroyed the forage as being an infectious material, you would have destroyed forage worth far more than the horses that had died! In fact, in not one case except the two I have quoted was there any more disease in the stable. It seems to me a very rash proceeding, unless you have more than one case of anthrax, to destroy the forage. I presume it is the food that carries the infection, and most frequently the oats; but I am unable to trace the method of infection in any of these cases. The rapid fatality and the danger to man give anthrax in a stable a more prominent position than other diseases, and the difficulty of tracing its origin adds to the anxiety of the owner.

HOG-CHOLERA OR SWINE-FEVER AND ITS ERADICATION.

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SINCE the discovery of a practical method of producing immunity against hog-cholera by Dorset and his co-workers, a new interest has become attached to this disease, and it is on account of this new interest that a discussion of its features is, perhaps, justifiable.

In order to condense our subject as much as possible within practical bounds, we will not make mention of the history of the disease and of the various phases of the evolution of the knowledge now on hand. We will also avoid discussion of the many points of purely scientific interest for the same reason.

There is, perhaps, no disease which has caused our farmers more palpable losses than this, while our scientists have for years exerted their best efforts in attempts to find a rational method of prevention. The earlier attempts of this nature were naturally all based upon the belief that Salmon's hog-cholera bacillus was the only specific cause of the disease, and while, no doubt, various investigators succeeded in rendering animals immune against this organism, it was apparent that a great deal of research work was still to be done before the solution of the problem could be looked for. The later work was begun by de Schweinitz, who had commenced to doubt the relation of the hog-cholera bacillus; and when, after the death of de Schweinitz, his ideas were tested and further worked out by his successor, Dorset, and his fellow-workers, the fact was established that hog-cholera is apparently due to an ultra-microscopic or filterable virus, and that if the *Bacillus suipestifer* plays a part at all in the etiology of the disease, it certainly is not a sole or primary one.

This discovery not only opened the way for the subsequent immunity experiments, but it also put a new phase on the disease known as swine-plague. This disease, most commonly associated with hog-cholera, is regarded as due to infection by *B. suisepticus*, but it is more probable that this organism also plays a secondary *rôle*.

In the outbreak among the swine of this institution, some of our cases showed lesions typical of swine-plague, others those of hog-cholera, and not a few those of both diseases. Both diseases, if such they be, were absolutely checked by the use of hog-cholera hyper-immune serum. I will not deny the pathogenic character of *B. suipestifer* and *suisepticus*; in fact, I believe that they define largely the patho-anatomic features of the disease; but it is my opinion, that for practical purposes, we may safely cease to make a distinction between the two types of disease.

As to the nature of and the part played by the so-called filterable virus, we do not know whether we have to deal with a true "contagium fluidum" or with an agent capable of either rendering the body specially vulnerable to infection, or of rendering certain micro-organisms specially aggressive.

Defibrinated hog-cholera blood, when injected into a susceptible animal, will promptly produce acute cholera, but it is probable that the natural infection takes place by way of the alimentary tract. In

such cases the virus is transmitted by the excretions of diseased animals and contaminated food and water.

As to the distribution of the disease from place to place, there are a number of well-known factors which play a prominent part, such as the congregation of swine from a great number of localities at stock shows, sales, &c. There are many instances in which such shows and sales have proved to be real clearing-houses of infection. A prominent part is also played by intermediary bearers, such as hog buyers, who travel far and wide from hog lot to hog lot; or neighbours, who, in a spirit of sympathy, visit the premises of friends whose hogs are dying with some fatal disease, and who carry infection on their shoes into their own hog lots. It is probable that animals such as birds, dogs, or coyotes, also are capable of carrying infection to considerable distance. The dust blown from infected lots also seems a very efficient distributor of infection. Food and drinking water may be contaminated, and thus serve as agents of dissemination. It is a well-known fact that hog-cholera has been carried from place to place by means of small streams.

The course of infection, when once introduced into a herd, varies, no doubt, with the virulence of the virus, or with its quantity. In some herds the hogs sicken in rather rapid succession, in others the evolution of the disease is slow. In most instances the progress of the infection is not very rapid, and such explosive outbreaks, as those we may see in anthrax, for example, are practically unknown in hog-cholera, yet in the individual animals the duration of actual disease may be exceedingly short.

The period of incubation also varies. After the hypodermic injection of small quantities of cholera blood, the first symptoms of sickness usually occur in from six to twelve days, but after natural exposure a much longer period often elapses. In some of our local outbreaks the disease made its appearance about one month after exposure.

The symptoms of cholera do not usually present a pathognomonic character. In the beginning of outbreaks peracute cases are seen. In those definite symptoms frequently fail, and the affected animal may be found dead, without having shown any previous indisposition.

In the acute form, which furnishes the greater percentage of our cases, lack of appetite and a disposition to separation from the rest of the herd is an early feature. The affected hog will bury itself in the litter, and is not easily induced to stir about. In white hogs we often find a darkening of the skin, sometimes to redness, and the bristles have a roughened appearance. There is redness, and often injection, of the visible mucosæ, while the body temperature is elevated.

Gastro-intestinal phenomena are an early feature of such cases. Vomiting is not uncommon, and diarrhœa is frequently seen, although it may be preceded by a constipation of variable duration. Diarrhœa and constipation not rarely are of alternate occurrence. The fæces in diarrhœa are liquid, of a light or dark yellow colour, and emit a fetid odour. In constipation the fæces are hard, the colour is dark or black, and they often show a covering of inspissated mucus, which in itself may be tinged with blood.

Respiratory difficulty is common. It varies from the symptoms expressive of catarrhal inflammation of the air-passages to those of a

high degree of dyspnœa, occasioned by pulmonary solidification and cardiac weakness.

The conjunctivæ are swollen, and the eyelids may be stuck together by a purulent exudate.

The skin in many cases shows an exanthema. On the abdomen, in the axillary, post-auricular, submaxillary and perineal regions, and on the inner side of the thighs a diffuse or circumscribed redness may be present, which later on may assume a purple tint. The skin so affected often shows blebs, which later on may dry up to black circumscribed scabs.

In the chronic forms of cholera the more prominent features are emaciation, digestive disturbances like an alternating diarrhœa, and constipation. There are commonly respiratory difficulties, as chronic pneumonia is a frequent complication. Such cases frequently show a fair appetite, and not rarely lead one to believe that recovery will take place. In the great majority of cases, however, the lesions present are sufficient to cause death.

Post-mortem appearances.—Since the elimination of the *B. suipestifer* the diagnosis of hog-cholera can no longer be rendered certain by bacteriologic tests, and we now have principally to depend on the pathoanatomic features to determine the identity of the disease. For this reason we undertake to sum them up in some detail, while in doing so we wish it to be understood that no attempt will be made in differentiating between the cholera and plague forms of the disease.

In a great number of cholera cases the skin presents lesions of an inflammatory nature. There may be seen a diffuse or circumscribed redness of the submaxillary and post-auricular regions. The same may be seen on the lower surface of the body, and is frequently most pronounced in the region of the axilla, the pubis, and the inner side of the thighs, as well as the perineum. In some cases the skin lesions consist of decided hæmorrhages, dark red in colour, now occurring as small punctiform hæmorrhages, then again as larger blotches. Pruritus is not uncommon.

As impetiginous exanthema is frequently seen in such cases in vesicles of varying sizes form, which are filled with a serous or sero-purulent fluid. After evacuation or absorption of the contents the vesicles dry up and become changed to a dark or black incrustation.

In other cases the skin lesions are in the nature of an urticaria, while during the recent outbreak the skin in one case markedly resembled the eruption seen in varicella.

Aside from the lesions mentioned, we frequently met with passive changes in the skin. These can be attributed to respiratory and circulatory difficulties, they are most commonly seen about the ears, and amount to a passive congestion, whereby the skin becomes dark in colour and more or less œdematous.

The lymph nodes are the organs which, as a rule, present the most constant lesions, and it is in those structures that the septicæmic character of the disease is most evident. In the beginning of outbreaks, when death from the peracute form of the disease is commonly seen, the lymph nodes are often the only organs showing a definite lesion, and in making the *post-mortem* diagnosis in cases of sudden death they must never be overlooked. In our autopsies during the recent outbreak we never failed to find these changes in the inguinal nodes, while, in fact,

they can be looked for in the other groups as well, although, perhaps, with less constancy.

One of the most common lesions is hyperplasia. The nodules are enlarged, greyish-white in colour, while their blood vessels are decidedly congested. They are frequently œdematous, and after a sufficient duration of the disease are apt to show necrotic foci.

In the peracute cases the lymph nodes more frequently present the picture of a hæmorrhagic lymphadenitis. The nodes are greatly swollen and are of a very dark colour, which may even be noticed through the fascia surrounding them.

In the chronic forms of cholera the lymph nodes are often found to be very firm on account of the proliferation of the connective tissue elements. In such organs secondary changes, such as hæmorrhages, softening, &c., may be met with. The mucosa of the mouth and pharynx are sometimes the seat of diphtheritic lesions, which may become changed to deeper ulcerations, according to the nature of the secondary infection which may be playing a part. A croupous-diphtheritic tonsillitis is very common, and when found should always arouse the suspicion of cholera.

In the more acute cases the mucosa of the stomach frequently shows a marked hæmorrhagic inflammation, which in more advanced cases may become complicated with diphtheritic lesions. The intestines are the seat of the most classic lesions of the disease, although the most typical ones are not a constant feature.

In a small portion of the cases there is a catarrh throughout the entire tract in which no erosions of the surface can be shown. The mucous membrane as well as the adenoid structures are swollen. This condition is not uncommon in the peracute form of the disease.

In quite a number of the peracute cases a hæmorrhagic enteritis is present without showing any diphtheritic lesions. In such cases the entire tract may be involved, although most commonly the lesions are most marked in the large intestines.

The lesions vary much in intensity; there may be diffuse redness, especially of the mucosa, but often also involving the other tunics. In addition to this redness, hæmorrhages in the mucosa, submucosa, and under the serous coat are common. They vary in size, from the mere punctiform specks to large blotches of the size of a half-dollar. They also vary much in colour, ranging from a bright red to a deep black, while in many instances I have seen a rusty colour.

In the severe peracute cases the entire intestinal wall is hæmorrhagically infiltrated, the wall itself being thickened and turgescent. In such cases the intestinal contents frequently have a liberal admixture of blood. Accompanying such lesions we find similar ones in the mesentery and meso-colon, while the corresponding lymph nodes are also hæmorrhagically infiltrated. The follicles of the intestinal mucosa may be the seat of inflammatory changes, giving rise to the anatomic picture of an enteritis follicularis.[1] In such a condition the follicles are markedly swollen, and are conspicuous as white round nodules. Their contents are softened and can be readily squeezed out. The intestinal lymphoid tissue is always hyperplastic. Ulceration of the involved follicles may lead to perforation.

[1] Poels, "Varkenssickten in Nederland."

The best known lesions of cholera are the intestinal ulcerations; even here many variations are seen. In one form we see a general confluent croupous-diphtheritic enteritis in which the mucosa is covered with a greyish-white croupous exudate. When this condition has existed for some days the intestinal wall becomes thickened and rigid, and the mucosa becomes necrotic and assumes a dark or black colour. In other cases the ulcerations are isolated, and vary from superficial erosions with a diphtheritic covering to deep excavations into the thickened intestinal wall. In some of such cases actual perforation takes place, causing death by shock and peritonitis on account of the escape of the contents. In other cases again there is an incomplete perforation, just sufficient for the escape of small amounts of intestinal contents. Such accidents are frequently followed by a plastic peritonitis, marked by extensive and firm adhesions, binding the intestinal mass into great conglomerations, which often can be found through the abdominal parietes of the living animal.

In some of the cases we meet with, and especially in the subacute and chronic ones, there may be evidence that the ulcerations are being repaired. In such cases a sharp line of demarcation differentiates the necrotic parts from the normal intestinal tissue. The croupous covering of ulcers becomes drier, shrinks up, and assumes a dark or black colour, while at the same time it is being pushed into the lumen of the gut by the proliferation of the normal tissues at the base of the ulcer. The characteristic button, thus formed, may become pedunculated and drop off, leaving a light-coloured cicatrix. In acutely fatal cases of cholera buttons are not often seen, as they are more essential to the chronic cases.

The spleen does not offer anything characteristic. There may be hemorrhages and petechiæ under the capsule, while in many cases nothing abnormal can be seen in this organ. In a number of cases the organ is considerably enlarged and of a black colour. In old chronic cases we may find necrotic foci in the spleen pulp.

The liver often shows a marked hyperæmia in the acute cases, and shows a parenchymatous degeneration in nearly all cases. In some of the more chronic forms of the disease this degeneration may be very complete, so as to justify the term "fatty liver." In such cases the organ is of a light yellow colour.

The peritoneum often participates in the disease. The lesions range from small hemorrhages to a real fibrinous or plastic peritonitis. In the latter cases the lesions are probably due to infection permitted to enter through lesions of the intestinal wall.

The kidneys show degenerative phenomena in all cases, and in some the kidney lesions remain confined to a more or less developed degeneration of the parenchyma. In the greater number of cases we find also evidence of hemorrhagic nephritis in the shape of a varying number of small punctiform hæmorrhages in the kidney substance and under its capsule. In one instance I found a very marked hæmorrhagic infiltration of the entire urinary tract, with an active subcapsular hæmorrhage of one of the kidneys.

The upper air-passages are frequently the seat of catarrhal conditions, while the lungs may present a variety of lesions, the majority of them probably being of a secondary nature.

Pulmonary congestion is very frequently found, as well as œdema,

while pneumonia is exceedingly common and is perhaps one of the most fatal complications in the chronic cases.

The inflammatory process may be lobar or lobular. In the former, usually confined to the anterior lobes, the line of demarcation between normal and inflamed lung is sometimes very sharp. The inflamed areas are usually of a light red, brownish, or grey colour. In the lobular form, inflamed lobules may be found scattered through the entire lung, and may be completely surrounded by intact lung tissue.

In cases of sometime standing necrotic changes are common, when necrotic foci spring up in the inflamed areas, The contents of these foci are cheesy or softened, while in some cases they are very similar to real abscesses. It is very probable that the patho-anatomic picture of cholera pneumonia is largely determined by the organisms which produce the secondary infection.

The pleura is frequently involved. This membrane, especially, its visceral portion, is often the seat of petechiæ, while intense adhesive pleurites are by no means uncommon.

The heart in the chronic cases commonly has a parboiled appearance, owing to fatty degeneration of the myocardium, while in the acute ones we frequently find hæmorrhages of varying size under the epicardium. The pericardium often shows hæmorrhages, while adhesive pericarditis is by no means uncommon.

That hog-cholera is not a disease which offers a field for curative intervention is a fact so well known that it will not be necessary to devote much space to the discussion of either treatment or prognosis. The mortality of the disease, in spite of the fact that for years the market has been glutted with cholera remedies, still continues to range between 80 per cent. and 100 per cent. Let us, therefore, turn to the more hopeful field of prophylaxis.

Prophylaxis.—Even this field, up to a year or so ago, offered but a very gloomy aspect. It is certain that the practice of isolation, segregation, disinfection, sanitation, &c., always had an inhibitive influence upon the spread of the disease, but their influence was not such that they could be relied upon as an absolute protection. For a long time it was apparent that if hog-cholera was to be eliminated it would have to be done by means of some process of immunization, and for many years science has occupied itself with this problem. It was not, however, until the discovery of the fact that the hog-cholera bacillus is not the primary cause of the disease that the way was opened to a practical solution of the immunization problem, because now experiments could be made in which the *B. suipestifer* played no part. As the result of the research made by Dorset, McBryde and Niles, it was found that hogs which had recovered from an attack of the disease possessed a very potent immunity, but that their blood could not be absolutely relied upon to confer immunity to other swine injected with it. When the immune hogs, however, were previously treated with large amounts of virulent blood, which they tolerate very well, their blood acquires a marked antitoxic property and confers a passive immunity to susceptible hogs injected with it.

The hyper-immune serum forms the base of the Dorset method of immunizing against hog-cholera. The method consists of rendering the animals passively immune, or, if an active immunity is desired, injecting them with a small quantity of virulent blood simultaneously,

or exposing them to natural infection while their immunity lasts. The immunity obtained after a serum injection lasts only about four weeks, but if this immunity is rendered active the animal will be protected for six months, probably much longer.

The serum is injected in doses ranging from 20 to 60 cc., according to the size of the animal. The injection is made subcutaneously on the inner side of the thigh. If virus is used it is injected in doses of 2 cc. simultaneously in the thigh of the opposite limb.

In some cases the immediate result is a small circumscribed swelling at the point of injection, and it is possible that this swelling is due to a local infection by the needle. It may also be due to the small amount of carbolic acid which has to be added to the serum in order to insure its keeping qualities.

The efficiency of this serum has been demonstrated in many instances, notwithstanding that failures have also to be recorded, and the question now presents itself as to the manner in which the Dorset method of immunization can be applied in order to secure the eradication of hog-cholera.

It seems certain that the use of the method can be depended on to eliminate hog-cholera as a source of serious loss to our swine-breeders, but if this has to be accomplished systematic action is necessary. The manufacture of the serum and virus should be under the control of the State live-stock sanitary authorities. These products should be placed at the disposal of swine-owners free of charge, or at cost, while their use, especially that of the virus, should be left to trained veterinarians operating under the direction of State authorities. The promiscuous use of the method by everybody will not have a general good result, while the sale of virus by manufacturing concerns should be prohibited, as it is not safe to place virulent material at the disposal of the general public.

It seems best to entrust the manufacturing of the serum to the State experiment stations, and to make it the duty of the State live-stock sanitary authorities to superintend its systematic application.

All cases of apparent infectious swine diseases should be promptly reported, and by methods of publicity swine-raisers should be educated to that effect. Such reports should at once be acted upon by a thorough investigation, and when death turns out to be due to cholera, immunization should at once be undertaken.

In the immunization of herds in which disease has occurred, it is probably better to use the simultaneous virus injection than to depend upon natural exposure. From the experience of the writer it seems, at least, that there are cases in which the infective material gains entrance to the exposed hogs so slowly, or in which the infectious material introduced is so slight, that either the passive immunity has already been used up before the introduction of infection, or that the amount of natural virus was too slight to produce an active immunity. The results in both cases are that an active immunity is not obtained, and that in course of time the animals sicken anyhow. The character of special strains of virus no doubt also plays a part. We, therefore, deem it best to immunize by simultaneous injection, so that we make certain of actual exposure.

The injections should be confined to healthy hogs only. The serum has no curative properties, and the injection of sick hogs would

only mean a waste of material. Furthermore, it would be a menace to permit sick hogs to continue their existence for a while longer, which again would mean an additional amount of infective material to be scattered far and wide. It is our opinion that, after the immunizing of healthy hogs, all sick ones should be immediately destroyed and burned, and if, in spite of immunization, some hogs should develop cholera, they should be disposed of in a similar manner.

This measure is not designed for the protection of the herd immunized, but simply to prevent infectious material being scattered over a community. In case immunization is done immediately after the occurrence of the disease, it may be sufficient to confine action to this one herd, unless other swine herds are close by, when it would be better to immunize all herds within a radius of half a mile.

When the infection has already made considerable headway in one or more herds, it seems advisable to include in the immunizing process all hogs within a radius of three or four miles, in order to surround the original focus of infection with a zone of immune animals. We also consider it of advantage to have the veterinarian who makes the injection prepare the virus to be used with the serum, whenever such is possible. The reason for preferring this procedure lies in the fact that in our experience we encountered some difficulty in preventing putrid changes in stored virus, no matter how carefully prepared and stored. Putrefaction does not seem to destroy the virulence of the blood, nor have we complaints about the virus issued, but on general principles we should prefer the fresh virus. This virus can be prepared readily, by the veterinarian in charge of the immunizing operations, from sick hogs found in the outbreak. Such animals are best fitted for this purpose when they are at their sickest. The necessary appliances could be furnished in a sterile condition by the experiment station supplying the serum. When no sick hogs are available, the virus stored previously should be used.

All this kind of work should be done at the State's expense, as it is done with a view of general protection, and as under such an arrangement immunization can be made more universal. Furthermore, local veterinarians in general practice, who should, in order to assure prompt action, be employed for this work as much as possible, should be given opportunity to familiarize themselve as much as possible with the *post-mortem* diagnosis of the disease, the prepartion of the virus, and the method in general. This should be arranged for at the serum plant of the experiment station, and at the State's expense. In addition to the measures here recommended, arrangements should be made by the managements of stock shows, sales, &c., with the live-stock sanitary authorities to have all swine exhibited properly immunized, so as to eliminate such institutions as possible disseminators of infection. The ordinary methods of quarantine, disinfection, and proper disposal of carcasses should also be adhered to.

TESTS OF BRUSCHETTINI'S HOG CHOLERA VACCINE AND BRUSCHETTINI'S HOG CHOLERA AND SWINE PLAGUE SERUM.[1]

THE act of Congress making appropriations for the Department of Agriculture for the fiscal year ending June 30, 1909, provides as follows:—

"That the Secretary of Agriculture is authorized to purchase in the open market samples of all tuberculin, serums, antitoxins, or analogous products, of foreign or domestic manufacture, which are sold in the United States for the detection, prevention, treatment, or cure of diseases of domestic animals, to test the same and to publish the results of said tests in such manner as he may deem best * * *."

In conformity with these provisions, samples of the following products have been tested by the Department:—

(1) Bruschettini's Hog Cholera Vaccine.

(2) Bruschettini's Hog Cholera and Swine Plague Serum.

Both of the products are distributed by the Sorby Vaccine Company (Inc.), 163, Randolph Street, Chicago, Ill.

(1) BRUSCHETTINI'S HOG CHOLERA VACCINE.

Two forms of labels for this product are known to be used. One reads in part, "Hog Cholera and Swine Plague Vaccine, Bruschettini." The other label is in Italian and French, and reads: "Vaccino contro il colera dei suini (vaccin anticolérique des porcs) Bruschettini."

In testing this product the directions for use furnished by the distributors were carefully followed. The test was made by injecting healthy pigs with Bruschettini's hog cholera vaccine and, after the lapse of ten days, placing these pigs in pens with hogs affected with hog cholera. All of the treated hogs remained well until exposed to disease in this way. After this exposure they all contracted the disease within the usual time, exhibiting typical symptoms, and all finally died, showing at autopsy typical lesions of hog cholera.

(2) BRUSCHETTINI'S HOG CHOLERA AND SWINE PLAGUE SERUM.

The labels on this product are in English and German, and read: "Serum Bruschettini against Hog Cholera and Swine Fever," and "Bruschettini'sche Serum gegen Schweineseuche und Schweinepest."

This product was also used in the manner directed by the distributors. Healthy pigs were injected with the serum, and after twenty-four hours these were exposed to hog cholera by being placed in pens with hogs affected with that disease. All of the hogs treated with Bruschettini's serum contracted hog cholera within the usual period of time after exposure, and finally died, exhibiting typical lesions of hog cholera at autopsy.

These tests indicate that neither Bruschettini's Hog Cholera Vaccine nor Bruschettini's Hog Cholera and Swine Plague Serum are reliable agents for protecting hogs from hog cholera.

[1] Circular of the United States Department of Agriculture.

STRANGLES IN HORSES, WITH FOUR COMPLICATED CASES.[1]

BY J. MARTIN RICE, V.S., BOBCAYGEON, ONTARIO, CANADA.

STRANGLES, commonly called by the laity "distemper," is an infective equine disease generally affecting young horses, but may affect horses at any age. The infective agent is a streptococcus, which can be found by cover-glass preparations, stained with gentian-violet, methylene blue, or by Gram's method. The disease is rarely fatal in the ordinary form.

The streptococcus of strangles is, no doubt, the more frequent cause of abscess in the horse than any other organism, and it is generally understood, both clinically and bacteriologically, that it is a common cause of acute catarrh and other respiratory diseases, with or without pus formation. The disease presents itself in many forms, affecting the various lymphatic tracts and glands.

When an outbreak commences in the pharyngeal region, it is common for all susceptible animals in the same stable to become affected in a similar position. Certain outbreaks seem to affect some particular part, although in some it may vary a little.

Symptoms in Pharyngeal Abscess.—Dyspnœa is present, owing to the narrowing of the air passage, but will depend upon the size and development of the abscess. The respiration, as a rule, is roaring. Sometimes a slight attack develops into a severe one with great rapidity. In the subparotid form, as a rule, one side alone is affected. If both sides, then the two abscesses may communicate. The abscess in this situation matures very slowly, and any attempt to liberate the pus, which lies deep, very often fails, even though several days have elapsed since the swelling commenced. The danger of wounding the blood-vessels is great, and if surgical means are adopted, either Hilton's or Viborg's method, which are somewhat similar, should be preferred. Hilton's method is to incise the skin over the most dependent part and then pass a blunt instrument, if possible, through into the abscess cavity. A pair of closed dressing forceps are now passed into the abscess cavity, opened wide and withdrawn; in this way the opening is dilated. Viborg, after incising the skin, used the finger to force an opening. Very often pus was not found, but was followed by a copious discharge in twenty-four or forty-eight hours. Some incise the skin over the most prominent part, which lessens the resistance, and is not a bad plan, the pus flowing out in about forty-eight hours. Sometimes the pus did not break out where the skin was incised, but at a higher or lower point. It is said that the local destruction of tissue for the outlet of the pus is not due altogether to pressure from within, but to the action of the ferment, or ferments, which are produced by the pus bacteria, and which possess the power of dissolving albumen.

Fistulæ of Stenson's duct sometimes occur, due to the breaking down of walls of the duct, by being included in the area of suppuration. Sometimes these abcesses break into the pharynx, and the pus is swallowed or inhaled into the lungs, and pyæmic pneumonia or pulmonary abscesses may result; but these can also be produced by metastasis, in which case they are generally met with in both lungs in the miliary form, or one large abscess may exist.

[1] Read at the Annual Meeting of the Ontario Veterinary Association, December 23, 1908.

These complications may be the result of pyæmia. If these lesions are very large death is the result; but on the other hand, if small, recovery make take place with persistent treatment. Sometimes an animal has an ordinary attack of strangles in the submaxillary space; it breaks, discharges, and heals. After two or three weeks, sometimes longer, the animal is noticed to have colicky pains: the fæces are slimy and hard, appetite poor, very dull, and the temperature on the increase. This is a sign of internal (mesenteric) abscesses. Again, the animal may breathe heavily; pressure upon the sides, or turning round sharp produces pain, and the temperature and pulse will be found to be increasing. This is a sign of purulent pleuritis and, as a rule, is fatal.

There are several other positions where the abscess sometimes occurs, such as the prepectoral form which, as a rule, is situated very deep. The encephalic abscess will cause the patient to walk one-sided, due to pressure upon the brain. Sometimes the abscess occurs on the scrotum, which gives it the appearance, at first sight, of scrotal hernia. Cutaneous strangles occurs about the face, eyelids, &c., in multiple abscesses, which give the muzzle a large appearance; in fact, the abscess may occur on any part of the body.

The form of strangles for which the purpose of this paper is intended is the internal form, which has occupied my attention conconsiderably. The following cases I will describe:—

Case 1.—Filly, aged 12 months. Abscess in pharyngeal and subparotid regions; temperature 105° F.; appetite appeared good, but deglutition was performed with great difficulty; breathing stertorous.

History.—The filly had an abscess form in the submaxillary space, and it had burst and healed about two weeks previously. The throat again swelled, but this time higher up.

Diagnosis.—Strangles affecting the pharyngeal and subparotid regions, and probably the guttural pouches; internal organs were also affected, which was indicated by difficulty in turning around.

Prognosis.—Unfavourable.

Treatment.—The usual treatment was given. The animal died three days later.

A few hours before death she appeared to be very much better, and ate steamed oats and hay and drank water.

Post-mortem.—Abscess of parotid and pharyngeal regions and guttural pouches. Pharyngeal abscess had broken and entered the trachea and lungs. When the trachea was severed the pus flowed out. Purulent pleuritis was also present, and several pints of pus were found in the pleural cavity. Pericarditis and pneumonia also existed. There was an abscess upon the stomach holding about half a pint of creamy pus, also innumerable small abscesses along the lymphatics of the bowels.

Case 2.—Mare, aged 8; weight about 1,500 lb. (owner same as Case 1); suffered from strangles. The abscess formed in the submaxillary space, burst, and healed. The temperature ranged between 101° and 102° F., and she appeared to be getting along very well, when suddenly the temperature rose to 105° F. It was at this time that the owner asked me to call and see the animal.

Symptoms.—On my arrival the following symptoms were observed: Heart-beats, 125 per minute; appetite in abeyance; laboured respiration; slight colicky pains, and bowels costive; temperature 105·3° F.

Diagnosis.—Pyæmic pneumonia, probably with mesenteric abscesses.

Prognosis.—Unfavourable.

Treatment.—Raw linseed oil, Oii.; rectal injections to stimulate bowels. The oil was repeated, but no movement of bowels resulted. Nux vomica and other stimulants and diuretics were given. Patient died two and a half days later. The rope was being put in the mouth for the purpose of raising the head for drenching, when the mare pulled back and dropped dead. During the two and a half days the pulse ranged between 112 and 135 beats per minute; the temperature dropped to 104° F. just before death; and she also drank milk and appeared much better.

Post mortem.—Solidification of a portion of both lungs with small abscesses. Purulent peritonitis and multiple mesenteric abscesses, large and small, containing creamy pus. There was rupture of the stomach, probably due to the fall at the time of death. Kidneys were very pale.

These two animals both died the same night, and only six hours apart. The *post mortems* were both performed at the same time, and after they were finished the owner said he had seven more suffering from the same disease, and wanted me to examine them.

Examination.—All the remaining horses had submaxillary abscesses, which were nearly healed; some had fresh abscesses forming. The temperatures ranged between 102·5° and 104° F. Their appetites were diminished. The owner had been giving them a heroin mixture, which he got made up at the drug store, but they did not improve by it.

Treatment.—Sodium hyposulphite ʒiv., pot. nit. ʒss., three times per day, and calcium sulphide ʒiv. twice per day for two-year-olds. Two-thirds of these doses were given to yearlings. Their throats were blistered with biniodide of mercury ointment (1 to 4), and the abscesses were thoroughly disinfected with creolin solution (1 in 10), containing 2 per cent. of formalin. There were three more fresh cases in the stable. They all made a good recovery.

Case 3.—Colt, aged 2, suffering from colicky pains, constipated, appetite poor. Submaxillary abscess had formed, which had burst and healed. The above is the history of the case as given by the owner. I informed the owner of the possibility of internal abscesses, and that it would very likely prove fatal.

Treatment.—Physic drench, sodium hyposulphite ʒii., pot. nit. ʒiii., three times per day (the two last-named medicines were given in bulk). The owner came back in two weeks time and asked me to visit the colt, because he was not doing very well. He remarked that the physic acted just right, but he was constipated again. Upon enquiring, I learned that he had only given half the medicine in the two weeks, which, had it been given according to directions, would only have lasted four days.

Examination.—Eyes very pale, condition poor, appetite in abeyance. Rectal examination revealed constriction, due to the pressure of a large abscess. The colt had not passed fæces for two days, and the last that was passed had an odour of decaying tissue.

Prognosis.—Unfavourable.

Treatment after Visit.—Purging drench composed of a small dose of aloes and oil was given, also nux vomica and rectal injections, but they failed to move the bowels. Pilocarpine and eserine were given,

which also failed; arecoline was tried, and this failed also. The colt died two days later.

Post mortem.—An abscess about the size of a large football was found situated in the right pelvic and posterior abdominal region, and about fifty mesenteric abscesses varying in size from that of a pea to an egg. The internal organs had a very fœtid odour.

Case 4.—Colt, aged 2 months, had an abscess form in submaxillary space, which was lanced three weeks previous to my visit. For the last three days the bowels had not moved, but this day the colt seemed much better, although it had to be helped upon its feet—a necessary proceeding for over a week—and when up, walked with its head to one side. (The above are the words quoted by the owner.)

I informed the owner I did not think it was much use treating it, because it would probably not live very long, but he insisted upon me going out to see it. Upon my arrival at the farm I was informed that the colt had been dead about an hour. Its legs were stretched out as though it had been struggling to get upon its feet.

Post mortem.—Mucous membranes very pale, pus discharging from right ear, and forehead was bulged out. Upon incising the forehead a large quantity of pus escaped. The cranial covering was removed, and a large quantity of thin watery pus was found in the cranial cavity. The guttural pouches contained a quantity of caseated pus about the size of an egg in each pouch. The posterior portion of bowels was empty; the anterior portion contained fæces and linseed oil (the oil was given the night previous). All along the mesenteric lymphatics there was an enormous number of small abscesses about the size of large beans, which contained creamy pus. There was a small rupture at the termination of the pulmonary vein.

Remarks.—Probably the rupture of the pulmonary vein was caused by the struggling to gain its feet. Walking to one side was due to pressure upon the brain. Paleness of the mucous membrane was due to the inferior state of the blood produced by the streptococcic infection.

Summary.—The majority of the cases of mesenteric abscess are due, no doubt, to the germs being carried by the blood-stream, or lymphatic system, to parts were they become lodged in the capillaries, &c. (secondary infection). If the abscess is allowed to mature well before opening, and then thoroughly disinfected with strong creolin solution, it would happen less often. In cases where surgical means must be adopted in the early stage, such as when the animal is strangling or cannot drink, the above-mentioned methods should be adopted, and the opening well cauterized, which will prevent the germs from so readily passing into the circulation.

The constipation is no doubt, in many cases, due to the toxins diminishing peristalsis, and in other cases to pressure of the abscesses upon the nerves.

Sodium hyposulphite and calcium sulphide have, in the writer's practice, proved very valuable, and may perhaps act in several ways: (1) By either destroying or inhibiting the growth of bacteria; (2) by increasing the phagocytic power of the leucocytes; (3) by increasing the bacteriolytic power of the blood plasma, or all three actions combined. The sulphides must be given in sufficient quantity to produce saturation in order to be effective.

Clinical Articles.

A CASE OF HERMAPHRODITISM (HERMAPHRODITISMUS VERUS LATERALIS) IN *SUS SCROFA*.

BY B. F. KINGSBURY, DEPARTMENT OF HISTOLOGY AND EMBRYOLOGY, CORNELL UNIVERSITY.

THE following report is made on material submitted to me by Dr. W. L. Williams, of the New York State Veterinary College, and at his suggestion :—

The specimen, from a pig, aged 9 months, was received from an Inspector of the Bureau of Animal Industry at Milwaukee. The

T. Testicle.
P. Penis.
O. Ovary.
U.C. Uterine Cornua.

FIG. 1.—Photograph of the genital organs, from the ventral side. (Loaned by Dr. W. L. Williams.)

external genitals were those of a male. The penis was normal. The perineum, however, presented a well-developed ridge, suggesting the vulva of the female animal. The internal genital organs indicated strongly that it was a case of true hermaphroditism.

As shown in the photograph of the specimen (fig. 1), there is the usual small corpus uteri, with two relatively long and convoluted uterine horns. Dissection revealed a typical cervix and a vagina. Cornua, corpus, cervix, and vagina possessed a lumen. As implied above, there was no orificium vaginæ. The left cornu is prolonged into a Fallopian tube, which terminates in a small fimbria, which is attached to a body about the size of a small bean, shown by microscopical examination to be an ovary. No trace was found of a testis, epididymis, or vas deferens on the left side. Upon the right side, however, there is an evident testis about an inch in length, with an

FIG. 2.—Follicle with contained ovum, from the ovary (left genital gland). (Photomicrograph.)

apparently typical epididymis, and a vas deferens. The right uterine horn resembles that on the left side. Its Fallopian tube ends, however, in a diminutive blind sack which was closely applied to the caput epididymidis, with which on superficial examination it appeared to be continuous. This, however, was not found to be the case. No ovary was found on this side.

The microscopical examination of the suspected ovary on the left side was confirmatory of the diagnosis. The middle third or more of the organ was cut out, and serial sections made. In the portion so examined five follicles containing ova were found, one of them being well developed, with a cavity, cumulus, theca, and stratum granulosum. A photograph is submitted showing a young follicle (fig. 2). Numerous small nests of cells resembling follicle cells were also found, but definite recognizable ova in such nests were

absent. The stroma ovarii was of normal structure. There was no indication of testicular tissue in the sections.

A longitudinal segment of the testis was also cut out and sectioned, revealing the typical structure of a cryptorchid testis. The tubules were composed of a single layer of cells, whose inner ends were vacuolated. The interstitial cells were numerous and typical, as shown in the photograph (fig. 3).

Adopting the classification of Klebs, the anomaly would be a case of hermaphroditismus verus lateralis, and as such seemed to me worthy of being put on record. As far as I have been able to ascertain there are

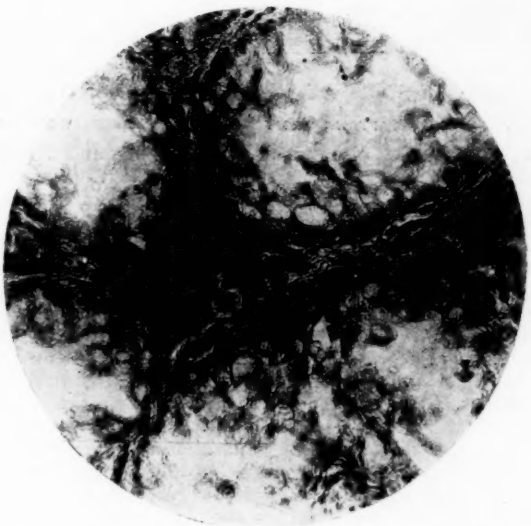

FIG. 3.—Section of cryptorchid (testis, right genital gland) showing the tubules and interstitial cells. (Photomicrograph.)

five other cases of true hermaphroditism recorded in the pig, in which the presence of ovary and testis were determined by microscopic examination; three cases in which ovary and testis were present on both sides, described by Garth, [1] and by Kopsch and Szymonowski [2]; one case, recorded by Pütz [3] of ovary and testis on one side only; a fifth case described by Duchanek [4] was inaccessible to me. More instances of hermaphroditismus verus are reported in the pig than in any other mammal save man, in whom seven cases are on record, the diagnosis being based on microscopic examination, and apparently authentic. Three of these are *H. unilteralis* (ovary and testis both present on one side only); one is *H. bilateralis* (Heppner's) (ovary and testis on both sides); while three are *H. lateralis* (ovary on one side, testis on the other).

It hardly need be said that no case in mammals is known of the presence of both ovarian and testicular tissue capable of functional activity other than possibly the elaboration of internal secretions.

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NOTES ON ACONITE POISONING.

BY R. C. MOORE, CAPTAIN, ARMY VETERINARY CORPS, WOOLWICH.

AFTER hearing the discussion on "Choking and Symptoms of Aconite Poisoning," at the last meeting of the Central Veterinary Medical Association, the following notes may be of interest :—

During the long and trying march over the Himalayas of the Tibet Mission Force to Lhassa, in 1904, many difficulties were encountered, and amongst them being the protection of transport animals from aconite poisoning. Several varieties of the plant—large and small—were noticed, all equally dangerous, growing sometimes over 6 ft. in height, and found in most parts of Tibet at an altitude ranging from 8,000 to 16,000 ft. Being forewarned, we went somewhat forearmed, as it was not only roadsides, grazing grounds and forage we had to fear, but also drinking places. The people of the country knew the action of the plant full well, so nothing was left to trust, and for this reason running water, whenever possible, was the order. Muzzling, clearing of roadsides, grazing grounds, and inspection of forage was closely attended to, but for all that numerous cases of poisoning occurred. The animals of the country, I have no doubt, left the plant wisely alone, for I never saw a case of aconite poisoning in a yak, Tibetan pony, goat, or donkey; and, moreover, I have seen these animals walking through the huge plants without tasting them. Mules and ponies from India and sheep bought from Bhutan suffered most.

The first symptom was smacking of the lips, frothing at the mouth, shortly to be followed by arching of the neck, and anti-peristaltic action of the œsophagus, similar to what is seen in a case of choking; staggering gait. Later, violent colicky pains, delirium, and death. The peculiar choking-like symptom was always present, and often accompanied by a rapid spasmodic contraction of the diaphragm.

Treatment consisted in the early administration of a hypodermic injection of physostigmine gr. i., atropine sulph. ½ gr., the former given about twenty minutes before the latter. This was found better than giving the two together, as the atropine seemed to delay the active purgative properties of physostigmine.

This treatment, if carried out in the early stages, was usually successful, but when colicky pains and delirium made their appearance nothing seemed to be of any use.

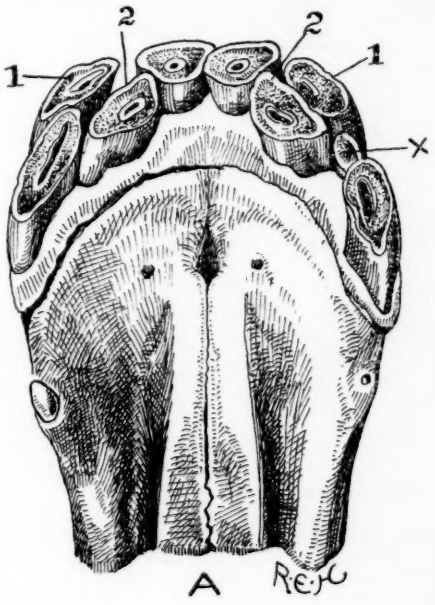
Post mortem generally showed gastro-enteritis, together with sickly odour, and frequently the leaves of the plant could be seen in the contents of the stomach.

The following is a fairly safe test for detecting aconite in drinking water and in the contents of the stomach: Slightly acidulate the suspected water or stomach contents with a few drops of acetic acid, and should aconite be present a red crystalline precipitate will be formed on the addition of a few drops of a solution of potassium permanganate.

THREE INTERESTING DENTAL VARIATIONS.

BY R. E. HOLDING.

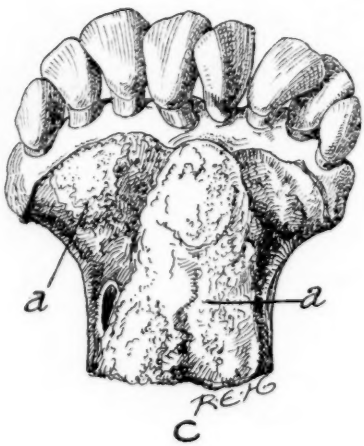
THAT the teeth of the domesticated animals are more liable to variation and congenital irregularity than their wild relatives is well known to the veterinary surgeon, and to the comparative odontologist and museum curator, through whose hands pass considerable collec-



tions of skulls belonging to different groups of wild animals, but who seldom comes across them. It is also a remarkable fact that these abnormalities occur most frequently in the animal which probably has been the longest under domestication—viz., the dog, for in the skulls

of the different breeds or varieties, from the long-headed Borzis to the almost spherical-headed Pekingese, every stage in the arrest or excess in the normal dental formulæ can be found. This is probably due to interference or interruption of the ordinary processes of development

B



C

by the specialist breeder; hence we get supernumerary legs, double-headed pigs, calves, and lambs, more or less united twins, and other abnormalities coming under the head of "Teratology."

A somewhat unusual case of supernumerary upper incisors in the

horse is illustrated in the drawing A, from a specimen I have recently obtained. It will be seen that in all there are nine incisors—viz., 2 being supernumerary and one, X, a retained deciduous tooth, whilst in the flesh it was difficult to decide which of the median incisors, 1 or 2, was the normal tooth; but upon dissecting off the gum and surrounding tissue it became apparent that, from their shortness, greater prominence on the outer side, and thinner alveolar walls, that No. 1 was the accessory or supernumerary tooth on each side. The mare was a heavy country-bred cart horse.

The incisors of the zebu (*Bos indicus*), the domesticated humped cattle of India (B), shows a somewhat rare variation—viz., the complete reversal of the position of the lateral incisor, *i.e.*, the inner face of the tooth becoming external. The animal was apparently aged 3½. There is a slight displacement of the corner tooth.

In the third specimen (C), from an ox obtained at a public slaughter-house, is of some pathological interest. The incisors are seen to be extremely irregular, and overlap, some being set well forward, others backward, not very well shown in a front view. It was pretty clear from an examination of the symphysis that at some early period of the animal's life, probably under 12 months, that a considerable fracture, with partial separation of the symphysis, had taken place, resulting in a bulbous mass of necrosed bone, with some periostitis and new growths at *a a*. The teeth were apparently of little use, and showed no wear at the edge.

A SUCCESSFUL HYSTEROTOMY ON A RUPTURED BITCH.

BY ARTHUR PAYNE, F.R.C.V.S., WEYBRIDGE.

THE subject was a Dandie Dinmont bitch, aged 5, operated on successfully by Professor Hobday, December, 1907, for left inguinal hernia.

About four weeks after the operation a small rupture became apparent on the opposite side, increasing in volume until, six months after, it assumed the proportions of the original, which was about the size of a goose egg.

On December 1, 1908, the bitch was brought to our surgery, as the day before and during œstrum she had escaped from custody and committed a *mésalliance* with a big fox-terrier. She was thoroughly douched with warm water, zinc sulph. 2 grains to the ounce being added. This proved unsuccessful, as eight weeks later she was brought in for her accouchement; a fœtus could be detected in the hernial sac.

When the period of gestation was accomplished we decided to perform a Cæsarean section. The seat of operation was prepared in the usual manner, and a general anæsthetic administered; instruments and silk (which was the only suture material used) boiled for five minutes.

A longitudinal incision was made, care being taken to avoid blood-vessels, and a few minutes allowed to elapse; the peritoneum opened with the aid of a director, and womb exposed. This was incised, and

fœtus and placenta removed. After swabbing out the uterus with boiled swabs, the walls were united with Lembert's bowel suture, one row only; an attempt was then made to return the organ to the abdominal cavity, but at the slightest attempt blood escaped between the sutures from the engorged uterus. The peritoneum was then brought together, and afterwards the skin, the seat of operation washed with boiled water, thoroughly dried, and sealed with collodion and iodoform. The skin wound had practically healed in ten days, excepting at one spot where there was a slight discharge, which continued for three or four days.

The patient never lost her appetite, and was cheerful from the moment she recovered from the effects of the chloroform. At one period during the operation respiration failed for some seconds, and artificial respiration was resorted to. Of course we were very lucky in finding only one pup; had there been more, graver risks would have been encountered.

No germicidal solution was used, but great care taken to render everything that had any relation to the operation thoroughly aseptic.

I hope to obtain the owner's consent to operate for reduction of the hernia at some future date.

HEART DISEASE IN A DOG.

BY R. F. STIRLING, M.R.C.V.S., GOVERNMENT VETERINARY DEPARTMENT, UMTALI, RHODESIA.

Subject.—A pointer dog, aged 4½ years; had never been worked, but was a lady's pet.

History.—When a pup had suffered from a very severe attack of distemper. The young lady, his mistress, told me that when she took the dogs for a run this particular one always became quickly fatigued and lagged behind. One morning he went out with some children, and after a few minutes' play lay down under a shrub and quickly died.

Post mortem.—All organs healthy except the heart. The edges of the cusps of the mitral valve were markedly thickened and shrunken, but there was no verrucose condition.

I think it is rather unusual to find this condition in a dog of this age.

Abstracts and Reports.

THIRD INTERIM REPORT OF THE ROYAL COMMISSION ON HUMAN AND ANIMAL TUBERCULOSIS.

AFTER the preamble the Report proceeds:—

Since the date of our Second Interim Report, the work of the Commission has been mainly directed to determining the special characters of the bacilli which are the cause of tuberculosis in animals other than the cow, and the relationships of the different types of tubercle bacilli which we have encountered in man and certain of the lower animals. The investigations bearing on these and other matters referred to in that Report as engaging our attention are not yet complete, and the publication of the results obtained must therefore be postponed. In the meantime we have thought it advisable to describe the results of a series of experiments which have been carried out by us with a view of obtaining information regarding the excretion or discharge of tubercle bacilli in the milk and fæces of tuberculous cattle.

In our Second Interim Report we expressed the opinion, as a result of our investigations, that a very considerable amount of disease and loss of life, especially among infants and children, must be attributed to the consumption of cows' milk containing tubercle bacilli.

Tuberculosis involving the udder is comparatively common in cows, and in such cases their milk always contains tubercle bacilli, and is therefore dangerous for human beings consuming it. It was, however, undecided what is the danger, if any, attaching to the milk of tuberculous cows in which the udder presents no evidence of disease. We therefore took the opportunity of making a number of observations and experiments bearing on this point. The experiments were made with the milk of cows which had contracted the disease in the natural way.

In natural tuberculosis in the cow, cases which show such obvious symptoms of the disease as emaciation and cough should be considered separately from the cases in which there are no such signs, and in which the disease is to be recognized during life only by means of the injection of tuberculin. None of the cows investigated showed any signs of disease of the udder during life, and in all, after slaughtering, the udder was carefully examined for tuberculous lesions and tubercle bacilli. No tuberculosis was found, except in one cow (cow F), in which one quarter of the udder showed four small nodules. These could not possibly have been detected during life.

We found that the milk of the cows obviously suffering from tuberculosis (see Appendix; cows B, C, and F) contained tubercle bacilli, whether the milk was obtained in the ordinary way or was withdrawn from the teat by means of a sterilized catheter. The presence of tubercle bacilli in the milk of cows clinically recognizable as tuberculous confirms the opinion we expressed in our Second Interim Report, that the milk of such cows must be considered dangerous for human beings.

The experiments which we have carried out with regard to the infectivity of the fæces of tuberculous cows were dictated by knowledge

of the fact that dirt of various kinds from cows and the cowshed is almost constantly present in milk as it reaches the consumer. Cows suffering from extensive tuberculosis of the lungs must discharge considerable numbers of bacilli from the air passages in the act of coughing, and some of the bacilli thus expelled may find their way into the milk. But our experiments indicate that the excrement of cows obviously suffering from tuberculosis of the lungs or alimentary canal must be regarded as much more dangerous than the matter discharged from the mouth or nostrils. We have found that even in the case of cows with slight tuberculous lesions, tubercle bacilli in small numbers are discharged in the fæces, while as regards cows clinically tuberculous, our experiments show that the fæces contain large numbers of living and virulent tubercle bacilli. The presence of tuberculous cows such as B, C, and F in company with healthy cows in the cowshed is therefore distinctly dangerous, as some of the tubercle bacilli which escape from their bodies in the excrement are almost certain to find their way into the milk.

The experiments are described in detail in the Appendix attached to this Report. They were carried out by Dr. F. Griffith, under our supervision, and we desire to express our high appreciation of the skill and care devoted by him to the work.

(Signed) W. H. Powell, *Chairman.*
G. Sims Woodhead.
Sidney Martin.
J. McFadyean.
Rubert Boyce.

APPENDIX.

(By Dr. F. Griffith.)

This Appendix gives the results of inoculation and feeding experiments made with the fæces and milk of naturally tuberculous cattle. Observations have been made on six milch cows. Three of the animals, B, C, and F, showed clinical evidence of tuberculosis, but in none during life could any tuberculous disease of the udder be detected. The object of this investigation was to ascertain if tubercle bacilli were present in the fæces and milk of these animals.

Description of Methods Used.—For the purposes of the experiments a considerable quantity of fæcal matter was required, and in order to avoid risk of contamination from the genito-urinary passages, the following method of collection was employed. The action of the rectum was stimulated by the injection into it of air through a sterile glass tube, and the fæces were received directly into a pail which was applied to the margin of the anal orifice. These precautions were necessary, as in one case, B, there was a purulent discharge from the vagina, and the *post-mortem* examination in three cases, B, C, and F, revealed extensive tuberculous disease of the uterus. A portion of the fæces was rubbed up in a mortar with sufficient salt solution to moisten it, and pressed through muslin to form an emulsion. This emulsion was used for the feeding and inoculation of guinea-pigs. The inoculation of fresh fæces into guinea-pigs frequently causes death within a few days, and it was possible to estimate roughly a minimal fatal dose

of the emulsion, which was prepared each day in the same way. It was found that 0·5 cc. of the emulsion almost invariably caused death from acute peritonitis, while after the inoculation of 0·05 cc. all the animals survived. The intraperitoneal method of inoculation was almost exclusively used.

The infectivity of the fæces was also tested by feeding guinea-pigs and swine. The swine were young animals, about 11 weeks old, belonging to three litters, and all obtained from the same farm. They were all tested with tuberculin before being used for experiment, and gave negative results in each case. Two animals were kept as controls, one from each of two litters, and when killed were found to be free from tuberculosis. No control to the third litter was kept, but all the eight pigs belonging to it though used for experiment were quite healthy when killed.

The milk of five of the cows, B, C, D, E, and F, was tested for the presence of tubercle bacilli, and strict precautions were taken to ensure absence of contamination during its collection. A metal catheter, connected by pressure tubing to a flask, was inserted into the milk sinus of the udder, a separate apparatus being used for each quarter, and the milk was withdrawn from the udder by the exhaustion of the air in the flask. Before the insertion of the catheter each teat was washed with a solution of perchloride of mercury and with methylated spirit, and the opening was inspected to ensure that no fæcal matter was pushed in with the catheter.

The milk thus obtained was tested as to the presence in it of tubercle bacilli by the inoculation of guinea-pigs, and occasionally of rabbits. Two guinea-pigs were inoculated intraperitoneally from each quarter, and each animal received when sufficient milk was obtained a dose of 10·0 cc. of uncentrifuged milk, plus the deposit of 20·0 cc. of centrifuged milk, or sometimes 6·0 cc. plus the deposit of 24·0 cc. In the case of three cows young swine were fed in each instance with the milk from the different quarters mixed together.

DETAILS OF THE EXPERIMENTS.

(1) Animals showing Clinical Signs of Disease.

Cow B.

This cow was a shorthorn, and had calved four weeks previously. It was in poor condition, and had a short infrequent cough; it reacted positively to the tuberculin test.

When killed it was found to have severe general tuberculosis. The lungs contained caseous masses which had broken down, and had become partly emptied with the formation of cavities. There were numerous tubercles along the whole length of the trachea, and the tonsils were infiltrated with breaking down caseous tissue. In the small intestine there were about sixty ulcers varying up to 1·5 cc, in diameter and scattered caseous nodules not yet ulcerated. The bronchial, mediastinal, mesenteric and portal glands were greatly enlarged and replaced by firm caseo-necrotic tissue. The posterior pharyngeal and the cervical glands were large and extensively caseated. The walls of the uterus were thick, firm, and infiltrated with tuberculous tissue, while the cavities were filled with muco-purulent

discharge such as issued from the vagina during life. The udder was free from tuberculous lesions.

The calf from this animal was in good condition and apparently healthy, but the *post-mortem* examination revealed disseminated tuberculosis. It was evident from the distribution of the disease (see *post mortem*), and its extent in relation to the age of the calf, that the tuberculosis had been acquired *in utero*. That the cow had been able to bear a calf is remarkable considering the advanced tuberculous disease of the uterus, though probably the pregnancy itself had hastened the progress of the disease.

Inoculation and Feeding Experiments with Fæces.—The fæces of Cow B passed on October 1, 3, 4, 5, 8, and 9, 1907, were tested for the presence of tubercle bacilli by the inoculation of guinea-pigs. In all twenty-five guinea-pigs were inoculated intraperitoneally, of which eight succumbed to acute infection in from one to two days. One, killed after twenty-seven days, was free from tuberculosis. The remaining sixteen were killed after about four weeks, and were found to be tuberculous—that is, all the guinea-pigs, with one exception, inoculated with fæces which lived for a sufficient period for tuberculosis to develop became tuberculous. The doses in relation to the amount of fæces passed were exceedingly small, consisting in the majority of cases of ·05 cc. of a thick filtered emulsion.

Guinea-pigs were also fed with the fæces. Six were fed from October 1 to October 9 inclusive with fæces mixed with sterilized milk, receiving no other food, and when killed were healthy. Two guinea-pigs were fed each day from October 1 to October 9, sixteen in all, with a single dose of fæces administered by means of a pipette, each receiving 1·0 cc. of the emulsion used for the intraperitoneal inoculations. They were killed after fourteen weeks, and were free from tuberculosis, in spite of the fact that they received fifty times the amount of fæcal emulsion which invariably caused tuberculosis when inoculated intraperitoneally.

Four young swine were fed with fæces mixed with sterilized milk from October 1 to October 9, 1907, the total amount shared between the four from the same trough being 19·1 kilogrammes. They were tested with tuberculin fifty-eight days after the experiment began, and all gave positive reactions, the rise of temperature varying in the individual cases from 1·3° C. to 3·0° C. They were killed after periods varying from sixty-three to one hundred days since the first feeding, and all showed generalized tuberculosis.

Inoculation and Feeding Experiments with Milk.—The milk of Cow B, collected in the manner previously described, was inoculated into guinea-pigs on October 11, 14, 15, 16, 17, 18, and 19, 1907. The inoculations were all made intraperitoneally, and two guinea-pigs were used for the milk of each quarter, each animal receiving a dose consisting of the deposit of 20·0 cc. of centrifuged milk, plus 10·0 cc. of uncentrifuged milk (except on the first day when each received the deposit of 15·0 cc.).

In all fifty-six guinea-pigs were inoculated. One was killed in seventy-nine days and had general tuberculosis without disease of the omentum. In these experiments I have not accepted tuberculosis in a guinea-pig as giving positive evidence of the presence of tubercle bacilli in the material inoculated unless there were lesions in the

omentum, muscle, or skin at the seat of inoculation containing tubercle bacilli, and therefore this guinea-pig was rejected. The remaining fifty-five guinea-pigs were killed, the majority after eleven weeks, and all were healthy with the exception of one which had tuberculosis, obviously due to the inoculation.

On November 5, 1907, the cow was inoculated with 20·0 cc. of tuberculin under the skin of the neck, after which she became ill, with laboured breathing. The next day, nineteen hours later, the milk was inoculated as before into eight guinea-pigs, of which four subsequently became tuberculous. The milk collected on November 7, forty-three hours after the inoculation of tuberculin, caused tuberculosis in seven out of the eight guinea-pigs inoculated.

Two young pigs were fed from October 14 to October 20 inclusive, receiving between them 18·4 litres of milk. They were killed eighty-six days after the first feeding and showed no tuberculosis. A third pig, which received on November 1 a single feed of 2·8 litres of milk, when killed sixty-nine days later was healthy.

The following experiments were performed in order to gain information as to the likelihood of the milk becoming infected during the ordinary process of milking.

On November 4 the cow was milked in the ordinary way, though with special precautions as to cleanliness, into an open pail; two out of four guinea-pigs inoculated with the mixed milk became tuberculous.

On November 5, before the tuberculin inoculation, the cow was milked without being cleansed in any way; two out of four guinea-pigs inoculated with the mixed milk became tuberculous.

The experiments are not conclusive, since it is possible that the tubercle bacilli were excreted in the milk, although use of it on seven separate occasions two or three weeks previously had caused tuberculosis in one guinea-pig only. In an animal so severely infected as this cow an invasion of the blood-stream by tubercle bacilli is always liable to occur, and these may be eliminated in the milk.

This natural distribution of tubercle bacilli in the course of the disease may account for the presence of tubercle bacilli in the milk after the inoculation of tuberculin, this inoculation being coincident with and not the cause of the dissemination.

Cow C.

This cow was a Jersey with obvious clinical signs of tuberculosis. She was emaciated and feeble, breathed hard, and had a frequent cough. Her temperature was febrile, and there was no rise sufficient to constitute a positive reaction after the inoculation of tuberculin. After death she was found to have severe thoracic tuberculosis; the lungs were closely beset with caseous nodules and large bright yellow caseo-necrotic masses, many of which were softening and breaking down. In several cases caseo-purulent substance was seen protruding from a necrotic mass into a bronchus, and the trachea was filled with a cast of inspissated muco-pus. The bronchial and mediastinal glands were greatly enlarged and caseo-necrotic throughout. The Peyer's patches of the small intestine all contained a few caseous nodules up to a hemp-seed in size, and there were several small ulcers up to 5·0 mm. in diameter. One mesenteric gland was greatly enlarged and

caseous. The uterus was extensively tuberculous, and the cavities were filled with purulent fluid and caseous flocculi. The udder was normal.

Inoculation and Feeding Experiments with Fæces.—The fæces of Cow C were tested for the presence of tubercle bacilli on October 24, 25, 28, 29, and 30, 1907, by the inoculation of thirty guinea-pigs. Of these, seventeen became tuberculous ; the rest died too soon for tuberculosis to develop.

Four guinea-pigs were fed continuously for six days with fæces mixed with sterilized milk ; one became tuberculous, the rest remained healthy.

Each of ten guinea-pigs received by the mouth from a pipette 1·0 cc. of the fæcal emulsion used for the inoculation experiments, and when killed after about ninety days all were found to be free from tuberculosis.

The following experiment gives some idea of the large numbers of tubercle bacilli in the fæces of this cow. One gramme of fæces was added to 2 litres of sterilized water, and two guinea-pigs were inoculated intraperitoneally, one with 1 cc., the other with 2 cc. of the mixture. Both developed severe general tuberculosis, the former dying in sixty-three days.

A young pig was fed with the fæces from October 23 to October 30 inclusive, receiving 5·56 kilogrammes mixed with sterilized milk. It was killed forty-eight days after the first feeding, and showed enlargement and caseation of the submaxillary and mesenteric glands, and disseminated tuberculosis.

Inoculation Experiments with Milk.—The milk was collected by catheter on each of three days, and was inoculated intraperitoneally into guinea-pigs and rabbits. Each guinea-pig received 10·0 cc. of uncentrifuged milk added to the deposit of 40·0 cc. of centrifuged milk.

None of the animals inoculated on October 25 became tuberculous. The milk of October 29 caused tuberculosis in two guinea-pigs, and that of October 30 caused tuberculosis in three rabbits and four guinea-pigs. The infectivity of the milk with reference to the individual quarters was irregular, but each quarter at some time gave tuberculous milk.

The explanation of this fact may be that the quantity of milk inoculated was not sufficient to be representative, rather than that the elimination of tubercle bacilli was irregular, since in several of the animals the slight amount of disease produced showed that only a few bacilli had been inoculated.

Cow F.

This animal was very ill : she was emaciated and feeble, and was kept under observation only for a few days, after which she was killed.

The *post-mortem* examination revealed severe general tuberculosis, apparently originating in the alimentary tract. In the left hind quarter of the udder, in the tissue forming the wall of the main sinus near the teat, there were four reddish-grey caseating nodules up to a pea in size. The rest of this quarter and the other three quarters were normal in appearance.

Inoculation Experiments with Fæces and Milk.—From the fæces of

Cow F, excreted January 29, 1908, seven guinea-pigs were inoculated intraperitoneally; of these four died too early for tuberculosis to have developed, and the remaining three, when killed after thirty-three days, had general tuberculosis.

The milk was collected on each of four days and inoculated into thirty-two guinea-pigs, of which eleven died prematurely. The remaining twenty-one developed general tuberculosis. Many of the guinea-pigs died in from eighteen to twenty-four days with tuberculosis resembling that following the intraperitoneal inoculation of 0·1 mg. of culture.

(2) Animals in which the only Evidence of Disease during Life was a Positive Reaction to Tuberculin.

Cow A.

This animal was a shorthorn heifer in good condition, and the diagnosis of tuberculosis depended entirely on the positive result of the tuberculin test.

When killed it was found to have on the right side an enlarged retro-pharyngeal gland which was cystic, with thick fibrous walls, and filled with breaking down caseo-necrotic substance. In the Peyer's patches of the small intestine there were three grey nodules containing caseous foci, two soft caseous nodules, and a small ulcer in which no tubercle bacilli could be demonstrated. There was also a caseous nodule in a mesenteric gland, caseation of a gluteal gland, and four tubercles in the lungs. All other organs and glands were healthy.

The fæces were collected for nine days from September 30, 1907, to October 9, 1907, and were used to inoculate and feed forty-one guinea-pigs, of which only one died prematurely. None of the guinea-pigs developed tuberculosis.

Four young swine were fed for the same length of time with the fæcal matter mixed with sterilized milk, the total amount shared between the four from the same trough being 25·6 kilogrammes of fæces. They were tested with tuberculin fifty-nine days after the experiment began, and one alone reacted, giving a rise of temperature of 2·6° C. This animal was killed a week later, and the *post-mortem* examination showed tuberculosis of the submaxillary glands on the left side, a caseous focus in one mesenteric gland, four caseating tubercles in the liver, and about thirty on the surface beneath the pleura of each lung. The remaining three swine were killed after ninety days, and were found to be free from tuberculosis.

Cow D.

This animal was fat and in good condition, and the diagnosis of tuberculosis rested entirely upon the positive result of the tuberculin test. At the *post-mortem* examination the lungs were found to contain discrete tuberculous nodules, several of which measured about 10·0 by 6·0 by 5·0 cm. On section, these were composed of softening yellow caseo-necrotic substance surrounded by fibrous walls. A few of the nodules had ulcerated into the bronchi, in which soft caseous substance was seen, and muco-pus could be squeezed out of many of the bronchioles. The bronchial and mediastinal glands were enlarged, and were to a large extent replaced by bright yellow, caseo-necrotic,

gritty masses. There was no tuberculosis of any other organ or gland in the body.

Inoculation and Feeding Experiments with Fæces.—The fæces of Cow D were tested on February 20 and 21 and March 24, 1908, by the inoculation of guinea-pigs and rabbits. Eleven guinea-pigs were inoculated intraperitoneally, of which two became tuberculous; two showed a few tubercles in the omentum containing moderately numerous acid-fast bacilli, and the remaining seven were free from tuberculosis. Two out of three rabbits inoculated developed tuberculosis. Two swine were fed for fourteen days with the fæces, and when killed after seventy-nine and ninety-nine days were found to be healthy.

Inoculation and Feeding Experiments with Milk.—The milk was collected by catheter on each of eight days, and was inoculated intraperitoneally into guinea-pigs; in all sixty guinea-pigs were used. None developed tuberculosis.

In several guinea-pigs the omentum contained a few translucent, sometimes whitish, foci, in which a few acid-fast bacilli were demonstrated in smear preparations. These bacilli were evidently dead, since guinea-pigs inoculated with the omentums did not develop tuberculosis, and only streptococci were isolated in cultures made from the foci.

Cow E.

The condition of this cow was very good; she gave a positive reaction to the tuberculin test. After death there were found in the lungs three small tuberculous nodules, and in the caudal mediastinal gland thirty or forty caseo-calcareous nodules up to a pea in size. In a suprarenal body there was a tubercle the size of a rape-seed, in which tubercle bacilli were demonstrated. There was no tuberculosis elsewhere.

Inoculation and Feeding Experiments with Fæces.—From the fæces passed on four days eighteen guinea-pigs were inoculated intraperitoneally. Of these five died (three prematurely), and the rest were killed after fifty days. All we free from tuberculosis. Two swine fed for fourteen days with the fæbes remained healthy.

Inoculation and Feeding Experiments with Milk.—The milk was collected by catheter on each of six days, and thirty-six guinea-pigs and four rabbits were inoculated intraperitoneally. None developed tuberculosis. Two swine were fed, each with 650·0 cc., and both remained healthy.

SUMMARY OF RESULTS.

The fæces of five naturally tuberculous cows, out of the total number of six so far investigated, have been found to contain living and virulent tubercle bacilli. Three of these animals, Cows B, C, and F, were severely diseased and were eliminating large numbers of tubercle bacilli; this is shown by the occurrence of tuberculosis after the inoculation of very small doses of fæcal matter in all but one of the guinea-pigs, which survived a sufficient length of time, and by the fact that all the swine fed became tuberculous. Two of the cows, A and D, were in apparently excellent condition of health. One cow, A, showed after death a caseous and cystic posterior pharyngeal gland, a few small nodules in the intestine, and slight disseminated

tuberculosis. The fæces of this animal caused tuberculosis in one out of four swine fed; the other three swine and all the guinea-pigs inoculated remained healthy. The other cow, D, had tuberculosis of the lungs, bronchial and mediastinal glands, without any disease elsewhere. The fæces of this animal caused tuberculosis in three guinea-pigs and two rabbits: two swine fed remained healthy.

The fæces of the sixth cow, E, which had slight tuberculosis of the lungs and a mediastinal gland, did not give rise to tuberculosis in any of the animals inoculated. Four cows, A, B, C, and F, out of the five cows which gave positive results showed some tuberculosis of the alimentary tract, but in at least one case, Cow C, it was not sufficient to account for the large numbers of tubercle bacilli in the fæces. These bacilli must have been coughed up from the lungs and swallowed.

Tuberculosis was present in the uterus of each of the severely infected cows, and the uterine discharge contained numerous tubercle bacilli. Such a condition constitutes another source of infection.

The milk of two of the cows, B and C, caused, though not invariably, tuberculosis in guinea-pigs inoculated with relatively small doses. The milk was withdrawn from the udder by catheterization, and *post-mortem* examination of the udders revealed no macroscopic evidence of tuberculosis. Small pieces were examined histologically by Dr. Eastwood, and we found to be normal, but it is of course impossible positively to exclude microscopical lesions. The milk of a third cow, F, caused severe tuberculosis in every guinea-pig which lived sufficient period of time after inoculation. The udder, except for four small nodules in the left hind quarter, was normal to the naked eye. The animal was very ill at the time the milk was collected.

The milk of the remaining two cows, D and E, tested did not give rise to tuberculosis in any of the animals inoculated.

[The Appendix contains full details of *post-mortem* examinations of all the animals, including cows, guinea-pigs, and swine. We reproduce the *post-mortem* record of one pig as being especially interesting and instructive as showing thoracic lesions from alimentary infection.]

Pig 210.

Fed with the fæces of Cow A from September 30, 1907, to October 9, 1907; Dose, one-fourth part of 25·65 kilogrammes of fæces. Age 11 weeks. Killed in good health, December 5, 1907 (sixty-six days after feeding began).

Weights.—September 30, 1907, 28 lb., December 5, 1907, 57 lb.; total gain in weight, 29 lb.

Tuberculin Tests.—September 16, 1907 (fourteen days before experiment began), reaction negative. November 28, 1907 (fifty-nine days after the experiment began), reaction positive. Rise of temperature 2·3° C.

Post-Mortem Examination.

General Condition.—Good.

Tongue, Tonsils, Larynx, and Pharynx.—Normal.

On the Left Side of the Neck extending from below the angle of the jaw to the root of the ear there was a chain of six glands, the largest

measuring 2·0 by 3·5 cm.; the smallest 1·6 by 1·2 cm. On section the largest consisted of a yellowish caseo-necrotic network in firm pinkish grey tissue. The other glands consisted of firm grey tissue with a fine yellowish network.

Right Submaxillary Glands.—Normal.

Cervical and Prescapular Glands.—Normal.

Abdomen.

Omentum and Peritoneum.—Normal.

Intestines.—Normal.

Mesenteric Glands.—The mesenteric glands were not enlarged, and in one a single caseous focus was seen.

Spleen.—Normal.

Liver.—On the convex surface of the liver there were four grey tubercles with opaque centres up to a rape seed in size. On the concave surface there were two. On section through the substance no more were seen.

Portal Glands appeared normal.

Kidneys.—In the cortices of the kidneys there were scattered grey points.

Suprarenal Bodies.—Normal.

Renal, Cœliac, Lumbar, and Iliac Lymphatic Glands.—Normal.

Thorax.

Lungs.—In the left lung on the surface just beneath the pleura there were thirty caseating tubercles varying up to a hemp seed in size; there was about the same number on the right side; they were sparsely scattered throughout the parenchyma.

Bronchial Glands.—An occasional irregular caseous focus could be seen in the bronchial glands.

Precrural and Inguinal Glands.—Normal.

Microscopical Examination (Smear preparations).

Kidney (grey foci).—Moderately numerous tubercle bacilli seen.

Mesenteric Gland (focus).—Eleven tubercle bacilli seen.

Bronchial Gland (focus).—No tubercle bacilli seen.

Reviews.

"The Food Inspector's Handbook." By Francis Vacher, Medical Officer of Health for Cheshire. Published by the Sanitary Publishing Co., Ltd. Price 7s. 6d.

Stimulated by the favourable reception with which four previous editions of this work have been received, and by the fact that the number of those who devote their whole time to meat inspecting is yearly on the increase, Dr. Vacher has revised and enlarged "The Food Inspector's Handbook," the fifth edition of which has just been placed on the market. The book relates the various statutory powers under which unwholesome food may be seized, and a consideration of the various abnormal conditions of foods which render them unfit for human consumption.

Dr. Vacher, like many other members of the medical profession, appears to favour the appointment of butchers as meat inspectors, although he is constrained to admit that "the ideal meat inspector must be a trained veterinarian." He endeavours to argue himself out of this position by making three assertions: (1) That (in this country) the system of meat inspection is not ideal. (2) That no self-respecting veterinary surgeon would apply for the post of meat inspector at the salary of £120 per annum. (3) That when in doubt the meat inspector can consult the Medical Officer of Health.

These statements are not actually made with the purpose of detracting from the value of the veterinary surgeon, but are calculated to support the claim that there is no reason why authorities should not get a man quite capable of performing such inspection as is attempted under existing conditions, without employing a veterinary surgeon, the substitute being referred to as "the best man they can get at the price."

"Existing conditions" are admittedly bad, and it should be our object to improve them. The fact that many veterinary surgeons would refrain from accepting the small salary offered by local authorities is a further indication of the necessity for the Government to establish a system of veterinary meat inspectors who would be properly paid. Although the average Medical Officer of Health may be able to judge in many cases as to the fitness of meat for human food, one cannot feel satisfied with a system of meat inspection run by the Medical Officer of Health and a butcher.

The Medical Officer of Health who does not number among his qualifications the diploma of the Royal College of Veterinary Surgeons has usually no very special knowledge of the diseases of animals.

A butcher in the position of meat inspector is, in many cases, compelled to act empirically. The suggestion that "a butcher is much more likely to know something of the tricks of his own trade," and is therefore likely to be a better meat inspector is, up to a certain point, apparently good argument; but in considering the qualifications of a food inspector, Dr. Vacher says "he must be honest." With that we entirely agree, but if the butcher, prior to obtaining his position as meat inspector, has been guilty of practising the "tricks of his trade" to the detriment of his customers, what guarantee have we that he will, in his new capacity, refrain from acting dishonestly?

An enumeration of the diseases of animals which render meat unfit for human food early in chapter v. is followed by a consideration of these conditions in chapter v. and chapter vi.

We do not consider these chapters to be of extraordinary merit, for in several places they are incorrect and out of date. Black quarter in bovines, for instance, is described as a form of anthrax. This, of course, is absolutely incorrect—anthrax and black quarter are two distinct diseases.

Anthrax in swine is referred to without mention being made to swollen throat as a symptom.

Texan fever is said to be allied to anthrax. The mistake undoubtedly arises from the fact that an enlarged spleen is often found in both these diseases. We know, however, that anthrax is caused by a bacillus, and that Texan fever is caused by a piroplasm.

The reference to braxy in sheep as splenic apoplexy is not good, for, in the first place, "splenic apoplexy" is not a term frequently applied to disease in sheep; and, secondly, because the term "splenic apoplexy" is associated with anthrax. It has not yet been proved that braxy is anthrax.

Joint-ill is said to be "probably always rheumatism." Rheumatism may be a useful term, but in a very large number of cases we know joint-ill arises from infection of the umbilicus.

Urticaria in swine is described separately from swine erysipelas. This may to a certain extent be justifiable, but probably the great majority of cases are due to the invasion of the animal body by the bacillus of swine erysipelas.

The chapters on poultry, game, fish, fruit, and vegetables contain very useful matter.

With the exception of the parts referring to the veterinary pathology above mentioned, we consider the book is likely to prove extremely useful, both to those engaged in food inspection and to those preparing themselves for examination in the subject.—M. J.

"Stable Management and Exercise."—By the late Captain Hayes, F.R.C.V.S. Published by Hurst and Blackett.

The continued popularity of Captain Hayes' books on horses is evidenced by the announcement of a second edition of his useful volume on "Stable Management and Exercise." The revision has been carefully carried out by the widow of the author, with the assistance of Colonel Steele, late of the Indian Army. The new edition will be published by Messrs. Hurst and Blackett about the 17th inst.

Spezielle Pathologie und Therapie der Haustiere.—(Special Pathology and Therapy of the Domesticated Animals). Vol. I. By Franz Hutyra and Dr. Joseph Marek. 1909.

This standard work, written in the German language by the two above-named Professors of the Veterinary School at Budapest in Hungary, is published by the firm of Gustav Fischer, of Jena. It is issued in two volumes of over 1,000 pages each, and the diseases included in the letterpress number 273, of which 92, the great majority of which are of an infectious nature, are comprised in the first volume. In the present publication Professor Hutyra, who deals

chiefly with the infectious diseases, has taken as his coadjutor Professor Marek, who, as his colleague, lectures on pathology and therapy at the Budapest School. The work and science gain by this co-operation, and the present issue is a considerable advance on the edition published in 1894-1898 in the Hungarian tongue by Hutyra.

Opening with a full account of anthrax, the differential bacterial diagnosis between this disease, malignant œdema, and quarter-ill is dwelt on, and the different methods of protective inoculation of five Continental authorities are explained. How man catches the disease by skin inoculation, inhalation, and ingestion, and the ways these infections declare themselves in the human subject, are adequately described.

The occurrence of malignant œdema is ascribed to wounds of the skin or mucous membranes, and in the latter case the reviewer thinks this is a point to be remembered in tooth extraction in all the domesticated animals.

Braxy in sheep is designated "gastromykosis ovis," and the bacillus which causes it is illustrated, and distinguished structurally from that of quarter-ill, which it resembles. The organism differs from that of the latter disease in being pathogenic for pigeons, fowls, and mice. There is a good illustration of pronounced swine erysipelas.

Hæmorrhagic septicæmias include those illnesses due to the bacillus *Bipolaris septicus*, and comprise fowl cholera, bovine, and ovine hæmorrhagic septicæmia, septic pleuro-pneumonia of calves, buffalo plague, "schweineseuche" (a name which the Germans limit to a pure lung disease of swine). Intravenous injections of collargol or ichthargan are recommended for navel-ill in foals when infection has become generalized. The pustular form of distemper (pasteurellosis) in the dog is noticed, but nothing is mentioned about the early appearance or diagnostic importance of a canine rash.

A sharp distinction is drawn between canine typhus (gastro-enteritis hæmorrhagica) and pasteurellosis of the dog, but the usefulness of chloretone or aphrodine in the former complaint is not indicated. The carrying out of good hygiene and nursing is given due prominence. Subcutaneous injections of trichloride of iodine and medicated inhalations are recommended. That the cow and dog can be affected with purpura hæmorrhagica is a fact not always recognized, but cases are described within the pages of the book.

Rinderpest occupies twenty-five pages. The appearances which we in this country associate with swine fever are given under "schweinepest." Notice is taken of the fact that *B. suipestifer* and *B. suisepticus* occur along with the filterable virus which occasions swine fever. The obligatory slaughter of diseased and suspected animals is insisted on, but protective inoculation and its performance against the disease are fully discussed, expediency travelling along with experimentation to try to reach the light.

Under acute exanthematous infectious diseases, cow-pox, horse-pox, and sheep-pox are described. The reviewer has never heard the name "grease disease" given to "horse-pox" in this country, and such a designation put down in the book as an English equivalent of the German "Pferdepocken" can only lead to confusion.[1]

[1] We believe that Jenner originally regarded horse-pox and grease as identical.—ED., *V.J.*

Doubt is expressed whether fowl diphtheria can be communicated to human beings, but some experimenters assert that they have found the fowl organisms in the throats of diphtheritic children.

Fowl-pox (*Epithelioma contagiosum avium*) would seem from the illustrations to cause terrible mischief in the throat, combs, and wattles of birds attacked. That fowls suffer from pox at all will doubtless be news to many.

Tetanus in the horse and dog is well illustrated, and also rabies. In the latter condition the peculiar frenzied look in the eyes of the canine example given as an illustrative picture is a credit to the illustrator.

Tuberculosis is treated in masterly fashion in 124 pages. All the matter is well up to date. The pseudo-tuberculosis of sheep, calves, and cattle is fully gone into. Actinomcyosis, botryomycosis, and glanders are copiously illustrated and written about.

A page is given to the appearances of glanders in man, and the authors write: "In many cases (of intestinal infection) the illness takes a very similar form to that of abdominal typhus, and only the persistence of fever over the third week and the occurrence of skin pustules clear up the cause."

Rickets is discussed under the heading "Rachitis," which ought to be altered. The authors advocate as therapy plenty of exercise in the open, and diet rich in lime. Bone meal and lime-water are recommended. The illustrations of a dog and a calf with rickets seem a bit overdrawn, but probably this is advantageous from a teacher's point of view.

Under affections of the circulatory organs, diseases of the pericardium, heart, and blood-vessels are done full justice to, and the chapters on aneurism, thrombosis, and embolism are well worthy of study. The French, Italian, and British synonyms for the German nomenclature are given at the commencement of each article, and the chapters on animal diseases as they affect human beings are very valuable and probably unique. The first volume is especially strong on preventive and curative inoculations, as can only be expected in a book of this kind, written in the home of such practices.

Vol. ii. will be reviewed later.—G.M.

Translations and Extracts.

SOME USES OF OXYGENATED WATER.

BY M. L. MAGNIN, VETERINARY SURGEON TO THE 5TH ENGINEERS, VERSAILLES.

NUMEROUS works have already been published on oxygenated water. Its employment tends to become more and more general in therapeutic veterinary surgery.

An article on the subject by M. Porcher in *Le Journal de Médecine vétérinaire et de Zootechnie*, and one by M. Quiclet in the sixth volume of the third series of *Recueil de Mémoires et Observations sur l'Hygiène et la Médecine vétérinaires militaires* are the most complete. The aim of this short note is simply to call the attention of my *confrères* to some applications of oxygenated water which I believe to be new.

Deep Firing.—M. Nocard, in *Le Bulletin de la Société centrale*, 1891, p. 430, calls attention to the excellent effects of antiseptics, and in particular powdered iodoformed ether to prevent sloughing of the skin after firing. Here, in oxygenated water, is a method of preservation far superior to the preceding. It has allowed and still permits me to fire deeply without distinction of season, that is to say it enables firing to be done during the coldest days of the year without any danger or serious inconvenience. For the rest, a little dexterity in the handling of the cautery, and certain hygienic measures, according to circumstances, are necessary; to place the animal operated on in a fresh and dark stable, and avoid exposure to the rays of a hot sun for any length of time. If discharge becomes too abundant take some hydrophile cotton and tamponize gently. Dab afterwards with oxygenated surgical water until the whole surface acted on is covered with a white foam; thoroughly drench the points which discharge most abundantly. Wait twenty to twenty-five minutes, then tamponize anew and dry the surfaces, and cover afterwards with iodoformed ether. Finally, this dressing should be applied twice during the day; once daily suffices afterwards.

Under the action of this treatment the itching disappears quickly, inflammation declines, too profuse discharge is lessened, and finally completely overcome. Oxygenated water destroys existing microbes; the swabbing penetrates the crevices. The iodoform opposes itself to the formation of new microbian colonies. The ether has for its end the favouring of the diffusion of the iodoform under the scales and into the depths of the wounds.

Collection in the Sinuses, Arthrites, Operations on the Foot and Neurotomies.—I have had occasion to employ pure oxygenated water in collections in the sinus, alternatively as a simple economic measure with other antiseptic solutions (permanganate of potash, sublimate and phenic acid), and the results have always been excellent. Under its action suppuration becomes less disagreeable to the smell, and less abundant.

In operations on the foot, complicated firing lines, treads, picked up nails, quittors, in the dressings necessitated by the accidents of shoeing, in neurotomies, pure oxygenated water, or diluted with its own volume of water, has constantly given far superior results to those

obtained with other antiseptics of more repute. Its only inconvenient effect is to give a yellow tint to the hairs of the region. In a case of traumatic arthritis of the hock with synovial discharge, and which finally took a grave turn, pure oxygenated water produced, at the end of six weeks, cessation of discharge, and complete disappearance of lameness. I will say in closing this short note that oxygenated water has never deceived me nor caused accidents. It is the most powerful antiseptic hitherto known. It is, in addition, an excellent disinfectant of the hands previous to a surgical operation.

(The translator can endorse all M. Magnin says about oxygenated water, and in addition, can say that when used warm it is an excellent remedy for purulent and catarrhal conjunctivitis.)—G. M.

(*Revue Générale de Médecine Vétérinaire.*)

A CASE OF SARCOSPORIDIOSIS OF THE HORSE.

(*Bul. de la Soc. cent. de Méd. Vét.* 1908.)

A MARE, ill for several months, presented at the head, neck, thorax, inferior abdominal wall, croup, thighs, and fetlocks, tumefactions comparable to the swellings of anasarca, or to cutaneous injuries due to boiling water. Flattened throughout their extent they have a superficies varying from the dimension of a nut to that of the "surface of the summit of a man's head." Their distribution is very unequal; isolated in certain regions, they are confluent in others. Under the belly and thorax they constitute a vast breastplate, extending from the girth to the sheath. The deformed head recalls that of animals with anasarca. These tumours are hard, ligneous, painless, of cartilaginous or calcareous consistence, adherent to the deep tissues. Movements of the animal are difficult, the illness appears like lumbago or muscular rheumatism. Functions normally performed. Prehension of food and mastication difficult. The tongue possesses all the characteristics of "wooden tongue."

Extirpation of a tumour reveals the psorospermic nature of the primitive lesion. Treatment by iodine produced no beneficial result. The sick animal suffers from rapid emaciation in spite of the abundant food he receives, and death soon occurs.

The autopsy shows that all the connective subcutaneous tissue, save in the region of the back and kidneys, is infiltrated, lardaceous, and weeps when scarified.

The panniculus carnosus muscle is fibrous throughout its extent. All the muscles are invaded in different degrees, the superficial ones being more gravely affected than the deep ones. In the first ones the lesions are unequally divided, the subcutaneous part and the inferior tendinous and aponeurotic extremities are chiefly altered. The pale muscles are much more affected than the red ones. The aspect of the lesions is equally varied; certain muscles entirely fibrous are microscopically free from parasitic lesions, others are studded with calcareous nodules in the middle of healthy tissue. The mode of infection remains unsuspected. Transmission experiments gave negative results.

(*Revue Générale de Médecine Vétérinaire.*)

ON THE SUPPRESSION OF BROKEN WIND.

BY SZÖLLÖS.

THE author, having learnt that certain gipsies and dealers in horses treat broken-winded horses with grains of datura stramonium, undertook a long series of experimental researches on this point at the Budapest Clinique.

The procedure of the gipsies and dealers is thus described by the author: The broken-winded horse is fed from seven to ten days with green forage or with dry forage, not dusty and softened with water; afterwards 25 to 45 grammes of datura grain mixed with the drinking water or with ½ pint of water is given; during the following half-day no food is given. Two hours after treatment the horse is ready for sale; he is exposed in a fresh place and protected from the wind. The action of the dosing commences after two hours, and is most marked at the end of four to eight hours, but may persist twenty-four hours. Dyspnœa always disappears when it is due to pulmonary emphysema; it is diminished when due to heart trouble, but never enough for a wide-awake man not to recognize it, especially after exercise prolonged for ten minutes.—Dr. MAREK (Budapest).—G. M.

(*Ex. Revue Générale de Médecine Vétérinaire.*)

A CASE OF PEMPHIGUS.

BY STAFF VETERINARY SURGEON KULL.

AT the beginning of March, 1908, an officer's horse, aged 8, fell ill, and exhibited the following appearances: In different parts of the body, on both sides of the neck, under the thorax, on the under surface of the lower jaw and neck, on the shoulders, the belly, the flanks, and the forearm, the upper and under thigh, the croup, the back, the loins, and sheath, as well as at the root of the tail, there were rows of raised blisters from the size of a walnut to the palm of one's hand, which were very sensitive. In places the rounded swellings were single and sharply defined, but for the most part they were confluent, widespread thickenings, and at the limbs they became rather large œdematous swellings. The skin at the affected parts was sensitive, and in the first few days itched a good deal, causing the patient to rub and bite himself.

On the second or third day the hair in the middle of some of the raised places fell out, and the skin at the same time became looser; the free-lying cutis vera was simply reddened, and appeared at certain places as if it had been punctured with a needle. There was a profuse exudation of yellow, clear, tenacious fluid, which hung in drops on the hair and dried into thick brown scabs. If the exuding skin was squeezed at this time, drops of serum welled up out of the pores, and the horse showed much pain.

After five days the places were dry, the scabs fell away, and the hairless spots were covered with thin transparent scales. Sensitiveness disappeared, and after a further four to six days the thickening of the dried parts of the skin declined. The hair now fell away and left large parts of the body hairless. New hair, however, soon replaced that which had fallen out. About three and a half weeks after the

first appearance of illness the horse was practically cured, and could be ridden again daily.

During the whole period of illness the rectal temperature only rose once for a few hours to 39·1° C. The pulse during the first three days was 50, and afterwards 40 to 44. Number of respirations, 10 to 12 per minute. There was loss of appetite the first six days of the illness, and the patient showed during this time a straddled gait with the hind limbs. There was no swelling of the lymphatic glands or vessels.

The treatment consisted in daily sponging with a 1 to 2 per cent. solution of lead acetate and alum. The hairless places were dressed with equal parts of tincture of aloes and glycerine, and internally sal. carolinum fact. and sodium bicarbonate were given. The food was clover, good hay, a few oats, and at times a carrot.

No reason can be given for the cause of the illness. It may have arisen from strong feeding with beans. Still, two other horses in the same stable received like food, but remained quite healthy.—G. M.

(*Zeitschrift für Veterinärkunde.*)

ON THE PRESENCE OF MICRO-ORGANISMS IN THE CONJUNCTIVAL FOLDS OF THE HORSE AND THEIR *RÔLE* IN AFFECTIONS OF THE EYE.

BY KRÜGER (*Zeitschrift für Veterinärkunde.*)

THE conjunctival folds give refuge to an important microbian flora, which finds in this medium extremely favourable conditions of culture. The introduction of micro-organisms occurs in different ways: they come from the atmosphere, where they are in suspension, and get deposited on the humid surface of the eye. They are particularly numerous in the air of stables, in the dust of forage and exercise grounds. Others arrive by the lachrymal ducts, which get infected at the level of their opening in the pituitary, always soiled; others finally occur from direct inoculation at the eyelids.

The author has studied this microbian flora, and obtained by a special method of culture the isolation of different species. The principal are: a staphylococcus, the *Streptococcus pyogenes* (aureus), a microbe in a case resembling the *Bacterium coli* another one identical with that of necrosis, and a diplococcus whose aspect and culture characteristics are those of the pneumococcus of Frænkel. For the most part the staphylococci and streptococci are pathogenic for mice; they cause an abscess at the point of inoculation, emaciation, and finally death. The diplococcus is always fatal.

The presence of these more or less pathogenic microbes in the conjunctiva of a healthy horse clears up for the most part the etiology of eye affections. The epithelial covering is in general a sufficient protection against the entrance of micro-organisms, but solutions of continuity are frequent, and constitute so many points of entry for micro-organisms. All catarrhal and purulent conjunctivites will thus be the result of the action of staphylococci and streptococci; Krüger attributes a particular pathogenic *rôle* to a pneumococcus, which is found in man, and causes specific ulcerations of the cornea. It would

be the determining cause of keratitis, following conjunctival inflammations, and which may lead to corneal perforation and infection of the aqueous humour lens and even of the eye (Troester); besides, a previous lesion of the conjunctival epithelium should not be always necessary for an infection that can be primarily produced.

Careful disinfection of the conjunctiva is of the highest importance in the treatment and prophylaxis of ocular affections. The author favours repeated baths with boric solution and antiseptic pomades, snch as:—

℞ Corrosive sublimate ... ... 0 gm. 003
White vaseline ... ... 10 gm.

which has the advantage of producing a protective veil on the eye.

Disinfection of the lachrymal ducts is excellent practice.

(*Revue Générale de Médecine Vétérinaire.*)

PROPOSED DEFINITION OF FALSIFICATION.

BY DR. HOTON (MALINES, BELGIUM).

Besides the principal task which the Congress of the White Cross took in hand in defining pure food stuffs, it seems to me that the different groups assembled at Geneva might come to some agreement as regards a definition of "falsification." Would it not be as well to replace by this term, which is that employed in the penal codes of Belgium and France, the different words which are sometimes substituted for it, such as adulteration, sophistication, fraud, alteration? The reports would gain in precision from the scientific, and, above all, from the judiciary, point of view, if an agreement was arrived at to adopt a single word, which ought to be well defined.

The correctional tribunal of Gand defined falsification as follows in 1888: "Punishable falsification consists in altering, changing, or denaturing a substance, be it by mingling or mixing, in introducing into it, or leaving in it, with fraudulent intent, a foreign substance, or even by extraction, taking from it fraudulently its natural qualities generally recognized in commerce."

One may ask if this definition is general enough to comprise the addition of antiseptics and colouring materials. To make this more complete it should have been drawn up as follows: "Punishable falsification consists in altering, changing, denaturing a substance, or by mixing or mingling, that is to say, by introducing into it, or leaving in it, a foreign substance with fraudulent intent; or by modifying its composition by extraction, that is to say, taking from it fraudulently its natural qualities generally recognized in commerce."

M. Depaire gave, in 1888, a definition of falsification which possesses the great advantage of conciseness and generalization: "Falsification is in principle all modification of the normal composition of a commodity destined for sale, unless the buyer is expressly forewarned." The word *denrée* (commodity) used by M. Depaire appears to me in a sense too limited. I would prefer to employ the word *substance* (substance) as of more general compass. Thus modified, Depaire's definition seems to me to give a perfect idea of falsification, and all countries might adopt it.—G. M.

(*L'Hygiène de la Viande et du Lait.*)

Correspondence.

To the Editors of the VETERINARY JOURNAL.

DEAR SIRS,—In your editorial remarks in this month's VETERINARY JOURNAL you suggest the imposition of an annual fee of a guinea and half-guinea, to be fixed by the Council every five years. As an opponent of this Bill against the guinea fee, I also make a suggestion, and hope some member of the Council will take it up. It is that the fees ought to be graduated on the income on the same lines as the income-tax.

A guinea may be of little account to a veterinary earning £500 or £600 a year in some of our big city practices, but to others situated like myself in a scattered country practice, where our gross income rarely exceeds £250 a year, I say it is a distinct hardship to be mulcted in a guinea every year. You ask the same from the man who struggles to maintain the dignity of the profession as far as his slender means will allow, as the man who has been fortunate in having an easy and lucrative practice. I throw out these hints hoping some abler pen may enlarge upon them.

A CONSTANT READER.

Books and Periodicals, &c., Received.

Journal of the Royal Army Medical Corps; Report of the Army Veterinary Service; London University Gazette; Board of Agriculture and Fisheries; Department of Agriculture and Technical Instruction for Ireland; Agricultural Journal of the Cape of Good Hope; Intossicazioni Alimentari da Carne (by Domenico Gualducci, Bologna); Sleeping Sickness Bureau; Wellcome's Laboratory Publications.

Letters and Communications, &c.

Dr. Van Es; W. L. Williams; B. F. Kingsbury; Captain Moore; Mr. Holding; Mr. Stirling; G. Mayall; "A Constant Reader"; A. W. Payne.

NOTE.—All communications should be addressed to 8, Henrietta Street, Covent Garden, London, W.C. Telephone, 4646 Gerrard. Telegrams, "Baillière, London."

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